Germano Melissano · Roberto Chiesa

AORTIC COMPLEXITIES

edi·ermes

Sample Chapters

Germano Melissano • Roberto Chiesa
Editors

AORTIC COMPLEXITIES

edi·ermes

AORTIC COMPLEXITIES
Germano Melissano • Roberto Chiesa (Editors)

ISBN 978-88-7051-663-0 - Paper edition

ISBN 978-88-7051-664-7 - Digital edition

Notices

Knowledge and best practice in this field are constantly changing. As new research and experience broaden our understanding, changes in research methods, professional practices, or medical treatment may become necessary.

Practitioners and researchers must always rely on their own experience and knowledge in evaluating and using any information, methods, compounds, or experiments described herein. In using such information or methods they should be mindful of their own safety and the safety of others, including parties for whom they have a professional responsibility.

With respect to any drug or pharmaceutical products identified, readers are advised to check the most current information provided (i) on procedures featured or (ii) by the manufacturer of each product to be administered, to verify the recommended dose or formula, the method and duration of administration, and contraindications.

It lies within the responsibility of practitioners, relying on their own experience and knowledge of their patients, to make diagnoses, to determine dosages and the best treatment for each individual patient and to take all appropriate safety precautions. To the fullest extent of the law, neither the Publisher nor the Authors, Contributors, or Editors, assume any liability for any injury and/or damage to persons or property as a matter of products liability, or arising from negligence or otherwise, or from any use or operation of any methods, products, instructions, or ideas contained in the material herein.

A book is the final product of a very complex series of operations that requires numerous tests on texts and images. It is almost impossible to publish a book with no errors. We will be grateful to those who find them and notify us. For enquiries or suggestions about this volume, please use the following address:

External relations - Edi.Ermes srl - Viale Enrico Forlanini, 65 - 20134 Milan (Italy)
Voice +39.02.70.21.121 - Fax +39.02.70.21.12.83

The Publisher is available for intellectual property owners with whom it was not possible to communicate, as well as for any inadvertent omissions and inaccuracies in the quotation of sources reproduced in this volume.

Paper edition printed in December 2018 by Faenza Printing Industries S.p.A. (RA), Italy
for Edi.Ermes - viale Enrico Forlanini, 65 - 20134 Milan, Italy
http://www.ediermes.it - Tel. +39.02.70.21.121 - Fax +39.02.70.21.12.83

Foreword

Treatments for aortic diseases have undergone explosive advances in the last decade. So too has our understanding of aortic disease processes. This progress in understanding and available treatments has come with the cost of increasing complexity.

Roberto Chiesa and Germano Melissano have edited a unique jewel of a book which deals with the many complexities of these advances in the pathogenesis of aortic diseases, their endovascular and open treatments and the management of treatment complications.

This volume, entitled Aortic Complexities, contains chapters by all those international innovators and thought leaders who have contributed the most to recent improvements in the management of aortic diseases. Accordingly it should be an invaluable up-to-date source of information for any physician or surgeon whose interests are focused on the aorta.

Drs. Chiesa and Melissano, who themselves are super stars in the aortic arena, have held a highly esteemed biannual International Vascular Congress which is focused largely, although not entirely, on the aorta and its treatment. This year's 2018 Congress, which will attract well over 1000 experts and delegates, is the eighth in the series and is entitled, "Aortic Surgery/ Peripheral & Venous: How to Do It". This volume on Aortic Complexities is published in conjunction with this 2018 Congress. Although the meeting will tell vascular surgeons, cardiac surgeons and others interested in these areas much about 'how to do it', it will also provide other valuable and relevant associated information. So too will Aortic Complexities in keeping with the favorably reviewed and widely referenced previous volumes comprising this outstanding series of books that deal with all aspects of the aorta, its diseases and their treatment.

Professor Chiesa and Professor Melissano are to be congratulated for bringing together the leading experts in the aortic arena to produce a text which provides a cutting-edge view of the current state of the art in aortic diseases and their treatment. Aortic Complexities will be a must-have volume for all libraries and for all those interested in the aorta, its newer treatments and associated complexities.

New York, December 2018

Frank J. Veith

Preface

Vascular Surgery, in particular Aortic Surgery, has undergone a radical revolution in recent years and, just as Dr. Veith forecast over two decades ago, endovascular approaches now play a leading role in the treatment of many vascular conditions.

Advanced technology and well-tested and standardized techniques now render the approach to an abdominal or thoracic aneurysm straightforward and relatively easy – when certain anatomic and pathologic features are present. However, there are a number of situations where the level of difficulty can rapidly shift from relatively easy to extremely challenging. These are what we call the "Aortic Complexities" that may include (but are not limited to): anatomy, the type of arterial disease, an underlying genetic condition, previous open or endovascular procedures, and specific clinical settings such as emergency or infection.

This book addresses the specific causes of complexity. These include diseases of the ascending aorta and the aortic root, open and endovascular solutions for thoracoabdominal aortic aneurysms, aortic dissection with its unique issues and new disease-specific techniques, ruptured aneurysms, organ protection, infection and genetic issues.

We are immensely grateful to our esteemed and distinguished colleagues who accepted the invitation to share with us their experience on aortic complexities in over fifty chapters that cover the whole range of aortic disease from the heart to the bifurcation of the abdominal aorta. The authors are all world renowned and leading experts, who took time off their busy schedules to contribute to this work. Reading their contributions was for us an exhilarating and highly instructive experience.

The publisher's (Raffaele Grandi) dedication to educational enterprises especially in the field of medicine and the precious and patient collaboration of Adriana Lombardi, the editorial assistant, were instrumental to the realization of the volume you are holding in your hands, and they deserve our full gratitude.

The first and final thoughts, while editing this volume, go to our aortic patients, our brave "aortic warriors" who endured the duress of aortic disease: to the survivors, and those who did not make it, and to their families and loved ones whose support is just as important as our surgical interventions for their outcome and wellbeing. In representation of all aortic survivors, we wish to thank our dear friend Timo Söderlund, founder, inspirer and motivator of the global movement of Aortic Disease Awareness. We hope that our combined efforts and determination will lead to a better understanding of this condition and help move a step forward in the fight against aortic disease.

Milan, December 2018

Germano Melissano *Roberto Chiesa*

Contributors

Aldo Alberti	San Giovanni di Dio Hospital, Florence, Italy
Vittorio Alberti	San Filippo Neri Hospital, Rome, Italy
Ottavio Alfieri	Scientific Institute Ospedale San Raffaele, Milan, Italy
Francesco Aloisi	San Camillo-Forlanini Hospital, Rome, Italy
Jean-Marc Alsac	Georges Pompidou European Hospital AP-HP, Paris, France
Ciro Amodio	S. Orsola-Malpighi Hospital, University of Bologna, Bologna, Italy
Michele Antonello	School of Medicine, University of Padua, Padua, Italy
Valeriy S. Arakelyan	Bakulev Scientific Center for Cardiovascular Surgery, Ministry of Health of Russian Federation, Moscow, Russia
Ferdinando Auricchio	Department of Civil Engineering and Architecture, University of Pavia, Pavia, Italy
Trissa Babrowski	University of Chicago Medicine, Chicago, IL, USA
Domenico Baccellieri	Vita-Salute San Raffaele University, Scientific Institute Ospedale San Raffaele, Milan, Italy
Marta Bargagna	Scientific Institute Ospedale San Raffaele, Milan, Italy
Gaia Barucco	Scientific Institute Ospedale San Raffaele, Milan, Italy
Filippo Benedetto	Policlinico G. Martino, University of Messina, Messina, Italy
Domenico Benevento	University of Siena, Siena, Italy
Tim Berger	University Heart Center Freiburg-Bad Krozingen, Freiburg, Germany
Luca Bertoglio	Scientific Institute Ospedale San Raffaele, Milan, Italy
Francesca Bianchi	Scientific Institute Ospedale San Raffaele, Milan, Italy

Guilherme Bicalho SITE-Serviço Integrado de Técnicas Endovasculares, Rio de Janeiro, Brazil

James H. Black III The Johns Hopkins Hospital, Baltimore, MD, USA

Carla Blanco Hospital Clínic of Barcelona, University of Barcelona, Barcelona, Spain

Leo A. Bockeria Bakulev Scientific Center for Cardiovascular Surgery, Ministry of Health of Russian Federation, Moscow, Russia

Cristina Botteri San Giovanni di Dio Hospital, Florence, Italy

Laura Capoccia Policlinico "Umberto I", Sapienza University of Rome, Rome, Italy

Heike Caravati Scientific Institute Ospedale San Raffaele, Milan, Italy

Gianpaolo Carrafiello Ospedale San Paolo, University of Milan, Milan, Italy

Patrizio Castelli School of Medicine, University of Insubria, Varese, Italy

Alessandro Castiglioni Scientific Institute Ospedale San Raffaele, Milan, Italy

Neal S. Cayne New York University-Langone Medical Center, New York, NY, USA

Maria Cristina Cervarolo School of Medicine, University of Insubria, Varese, Italy

Laurent Chiche Pitié-Salpêtrière Hôpital Universitaire, Paris, France

Roberto Chiesa Vita-Salute San Raffaele University, Scientific Institute Ospedale San Raffaele, Milan, Italy

Emiliano Chisci San Giovanni di Dio Hospital, Florence, Italy

Debra S.T. Chong The Royal Free London NHS Foundation Trust, London, United Kingdom

Marc Coggia Hôpital Ambroise Paré, Boulogne-Billancourt, France

Elda Chiara Colacchio Hôpital Ambroise Paré, Boulogne-Billancourt, France

Michele Conti Department of Civil Engineering and Architecture, University of Pavia, Pavia, Italy

Raphaël Coscas Hôpital Ambroise Paré, Boulogne-Billancourt, France

Joseph S. Coselli Baylor College of Medicine, Houston, TX, USA

Rodrigo Cunha SITE - Serviço Integrado de Técnicas Endovasculares, Rio de Janeiro, Brazil

Simone Cuozzo Policlinico "Umberto I", Sapienza University of Rome, Rome, Italy

Marco Cursi Scientific Institute Ospedale San Raffaele, Milan, Italy

Martin Czerny University Heart Center Freiburg-Bad Krozingen, Freiburg, Germany

Ronald L. Dalman Stanford University School of Medicine, Stanford, CA, USA

Jean-Michel Davaine Pitié-Salpêtrière Hôpital Universitaire, Paris, France

Gianmarco de Donato Department of Medicine, Surgery and Neuroscience, University of Siena, Siena, Italy

Monica De Luca Scientific Institute Ospedale San Raffaele, Milan, Italy

Eike Sebastian Debus University Hospital Hamburg-Eppendorf, Hamburg, Germany

Ubaldo Del Carro Scientific Institute Ospedale San Raffaele, Milan, Italy

Roberto Di Bartolomeo S. Orsola-Malpighi Hospital, University of Bologna, Bologna, Italy

Luca Di Marco S. Orsola-Malpighi Hospital, University of Bologna, Bologna, Italy

Holger Diener University Hospital Hamburg-Eppendorf Hamburg, Germany

Anahita Dua Stanford University School of Medicine, Stanford, CA, USA

Ejona Duka Ospedale San Paolo, University of Milan, Milan, Italy

Mohamed Hosni Eldossoki Cairo University, Cairo, Egypt

Gianluca Faggioli Policlinico S. Orsola, University of Bologna, Bologna, Italy

Aaron Fargion University of Florence, Florence, Italy

Elsa Madeleine Faure Georges Pompidou European Hospital AP-HP, Paris, France

Cecilia Fenelli Policlinico S. Orsola, University of Bologna, Bologna, Italy

Angela M.R. Ferrante Fondazione Policlinico Universitario A. Gemelli IRCCS, Università Cattolica del Sacro Cuore, Rome, Italy

Stefania Ferraro School of Medicine, University of Insubria, Varese, Italy

Diego Ferreira SITE - Serviço Integrado de Técnicas Endovasculares, Rio de Janeiro, Brazil

Marcelo Ferreira SITE - Serviço Integrado de Técnicas Endovasculares, Rio de Janeiro, Brazil

Alice Finotello IRCCS Ospedale Policlinico San Martino, University of Genoa, Genoa, Italy

Chiara Floridi Ospedale San Paolo, University of Milan, Milan, Italy

Marco Franchin School of Medicine, University of Insubria, Varese, Italy

Alessandro Frigiola IRCCS Policlinico San Donato, San Donato Milanese (Milan), Italy

Enrico Gallitto Policlinico S. Orsola, University of Bologna, Bologna, Italy

Mauro Gargiulo Policlinico S. Orsola, University of Bologna, Bologna, Italy

Valerio Gazzola IRCCS Ospedale Policlinico San Martino, University of Genoa, Genoa, Italy

Ilaria Giambuzzi Scientific Institute Ospedale San Raffaele, Milan, Italy

Olivier Goëau-Brissonnière Hôpital Ambroise Paré, Boulogne-Billancourt, France

Alexander Gombert European Vascular Center Aachen-Maastricht, University Hospital RWTH Aachen, Aachen, Germany

Alessandro Grandi Scientific Institute Ospedale San Raffaele, Milan, Italy

Viviana Grassi Fondazione Ca' Granda - Ospedale Maggiore Policlinico, University of Milan, Milan, Italy

Franco Grego School of Medicine, University of Padua, Pauda, Italy

Stephan Haulon Hôpital Marie Lannelongue, Université Paris Sud, Le Plessis Robinson, France

Franziska Heidemann Munich University Hospital, Munich, Germany

Caitlin W. Hicks The Johns Hopkins Hospital, Baltimore, MD, USA

Michael J. Jacobs European Vascular Center Aachen-Maastricht, University Hospital RWTH Aachen, Aachen, Germany

Heinz Jakob University Hospital of Essen, Essen, Germany

Isabelle Javerliat Hôpital Ambroise Paré, Boulogne-Billancourt, France

Andrea L. Kahlberg Vita-Salute San Raffaele University, Scientific Institute Ospedale San Raffaele, Milan, Italy

Jussi M. Kärkkäinen Mayo Clinic, Rochester, MN, USA

Mahine Kashi Pitié-Salpêtrière Hôpital Universitaire, Paris, France

Piotr M. Kasprzak University Hospital Regensburg, Regensburg, Germany

Athanasios Katsargyris Paracelsus Medical University, Nuremberg, Germany

Maria Katsarou Scientific Institute Ospedale San Raffaele, Milan, Italy

Mary Bo Kyung Kim Baylor College of Medicine, Houston, TX, USA

Tilo Kölbel University Hospital Hamburg-Eppendorf, Hamburg, Germany

Nikolas Konstantinou Munich University Hospital, Munich, Germany

Drosos Kotelis European Vascular Center Aachen-Maastricht, University Hospital RWTH Aachen, Aachen, Germany

A. Mohammed Idhrees SIMS Hospitals, Vadapalani, Chennai, India

Ali Irshad Houston Methodist Hospital, Houston, TX, TUSA

Mario L. Lachat Zurich University Hospital, Zurich, Switzerland

Alex Larena-Avellaneda University Medical Center Hamburg-Eppendorf, Hamburg, Germany

Alice Le Huu Baylor College of Medicine, Houston, TX, USA

Alessandro Leone S. Orsola-Malpighi Hospital, University of Bologna, Bologna, Italy

Marco Leopardi San Salvatore Hospital, University of L'Aquila, L'Aquila, Italy

Mauro Lo Rito IRCCS Policlinico San Donato, San Donato Milanese (Milan), Italy

Patrizia Lo Sapio San Giovanni di Dio Hospital, Florence, Italy

Diletta Loschi Scientific Institute Ospedale San Raffaele, Milan, Italy

Alan B. Lumsden Houston Methodist Hospital, Houston, TX, USA

Vladimir Makaloski University Hospital Hamburg-Eppendorf, Hamburg, Germany

Michel S. Makaroun University of Pittsburgh Medical Center, Pittsburgh, PA, USA

Simone Mambrini IRCCS Ospedale Policlinico San Martino, University of Genoa, Genoa, Italy

Nicola Mangialardi San Camillo-Forlanini Hospital, Rome, Italy

Kevin Mani Uppsala University, Uppsala, Sweden

Armando Mansilha Centro Hospitalar São João, Porto, Portugal

Wassim Mansour Policlinico "Umberto I", Sapienza University of Rome, Rome, Italy

Rossella Marcucci University of Florence, Florence, Italy

Mario Marino San Camillo-Forlanini Hospital, Rome, Italy

Pablo Marques de Marino Paracelsus Medical University, Nuremberg, Germany

Daniele Mascia Scientific Institute Ospedale San Raffaele, Milan, Italy

Fabrizio Masciello University of Florence, Florence, Italy

Chiara Mascoli Policlinico S. Orsola, University of Bologna, Bologna, Italy

Tara M. Mastracci The Royal Free London NHS Foundation Trust, London, United Kingdom

Cristina Mattioli Scientific Institute Ospedale San Raffaele, Milan, Italy

Mariagnese Mele University of Siena, Siena, Italy

Germano Melissano Vita-Salute San Raffaele University, Scientific Institute Ospedale San Raffaele, Milan, Italy

Gaspar Mestres Hospital Clínic of Barcelona, University of Barcelona, Barcelona, Spain

Stefano Michelagnoli San Giovanni di Dio Hospital, Florence, Italy

Ross Milner University of Chicago Medicine, Chicago, IL, USA

Abhisekh Mohapatra University of Pittsburgh Medical Center, Pittsburgh, PA, USA

Fabrizio Monaco Scientific Institute Ospedale San Raffaele, Milan, Italy

Giacomo Murana S. Orsola-Malpighi Hospital, University of Bologna, Bologna, Italy

Pasquale Nardelli Scientific Institute Ospedale San Raffaele, Milan, Italy

Fabrizio Nesi San Camillo-Forlanini Hospital, Rome, Italy

Gustavo S. Oderich Mayo Clinic, Rochester, MN, USA

Kyriakos Oikonomou University Hospital Regensburg, Regensburg, Germany

José Oliveira Pinto Centro Hospitalar São João, Porto, Portugal

Matteo Orrico San Camillo-Forlanini Hospital, Rome, Italy

Davide Pacini S. Orsola-Malpighi Hospital, University of Bologna, Bologna, Italy

Giancarlo Palasciano University of Siena, Siena, Italy

Domenico Palombo IRCCS Ospedale Policlinico San Martino, University of Genoa, Genoa, Italy

Bianca Pane IRCCS Ospedale Policlinico San Martino, University of Genoa, Genoa, Italy

Claudia Panzano University of Siena, Siena, Italy

Edoardo Pasqui University of Siena, Siena, Italy

Karin Pfister University Hospital Regensburg, Regensburg, Germany

Michele Piazza School of Medicine, University of Padua, Padua, Italy

Gabriele Piffaretti School of Medicine, University of Insubria, Varese, Italy

Rodolfo Pini Policlinico S. Orsola, University of Bologna, Bologna, Italy

Janet T. Powell Imperial College London, London, United Kingdom

Carlo Pratesi University of Florence, Florence, Italy

Giovanni Pratesi University of Rome Tor Vergata, Rome, Italy

Zoran Rancic University Hospital Zurich, Zurich, Switzerland

Paola Redaelli Scientific Institute Ospedale San Raffaele, Milan, Italy

Vincent Riambau Hospital Clínic of Barcelona, University of Barcelona, Barcelona, Spain

Eduardo Rodrigues SITE - Serviço Integrado de Técnicas Endovasculares, Rio de Janeiro, Brazil

Fiona Rohlffs University Hospital Hamburg-Eppendorf, Hamburg, Germany

Sonia Ronchey San Filippo Neri Hospital, Rome, Italy

Giulia Rossi Pitié-Salpêtrière Hôpital Universitaire, Paris, France

Wilma Schierling University Hospital Regensburg, Regensburg, Germany

Carlo Setacci University of Siena, Siena, Italy

Francesco Setacci IRCCS Multimedica Milan, Italy

Michael J. Singh University of Pittsburgh Medical Center, Pittsburgh, PA, USA

Pasqualino Sirignano Policlinico "Umberto I", Sapienza University of Rome, Rome, Italy

Jonathan Sobocinski Centre Hospitalier Régional Universitaire de Lille, Lille, France

Timo Söderlund Sandared, Sweden

Karl Sörelius Uppsala University, Uppsala, Sweden

Francesco Speziale Policlinico "Umberto I", Sapienza University of Rome, Rome, Italy

Sara Speziali University of Florence, Florence, Italy

Giovanni Spinella IRCCS Ospedale Policlinico San Martino, University of Genoa, Genoa, Italy

Domenico Spinelli Policlinico G. Martino, University of Messina, Messina, Italy

Francesco Squizzato School of Medicine, University of Padua, Padua, Italy

Benjamin W. Starnes University of Washington, Seattle, WA, USA

Andrea Stella Policlinico S. Orsola, University of Bologna, Bologna, Italy

James J. Suffoletta Rice University, Houston, TX, USA

Antonino Tarallo School of Medicine, University of Insubria, Varese, Italy

Emanuel R. Tenorio Mayo Clinic, Rochester, MN, USA

Matteo Tozzi School of Medicine, University of Insubria, Varese, Italy

Tom Treasure University College London, London, United Kingdom

Santi Trimarchi Fondazione Ca' Granda - Ospedale Maggiore Policlinico, University of Milan, Milan, Italy

Konstantinos Tsagakis University Hospital of Essen, Essen, Germany

Yamume Tshomba Fondazione Policlinico Universitario A. Gemelli IRCCS, Università Cattolica del Sacro Cuore, Rome, Italy

Nikolaos Tsilimparis University Hospital of the Ludwig-Maximilian University, Munich, Germany

Isabelle Van Herzeele Ghent University Hospital, Ghent, Belgium

Alessandro Varrica IRCCS Policlinico San Donato, San Donato Milanese (Milan), Italy

Frank J. Veith New York University-Langone Medical Center, New York, NY, USA

Bashi V. Velayudhan SIMS Hospitals, Vadapalani, Chennai, India

Eric L.G. Verhoeven Paracelsus Medical University, Nuremberg, Germany

Alessandro Verzini Scientific Institute Ospedale San Raffaele, Milan, Italy

Arno von Ristow Pontifical Catholic University, Rio de Janeiro, Brazil

Alessio Vona San Camillo-Forlanini Hospital, Rome, Italy

Anders Wanhainen Uppsala University, Uppsala, Sweden

Sabine H. Wipper University Medical Center Hamburg-Eppendorf, Hamburg, Germany

Xavier Yugueros Hospital Clínic of Barcelona, University of Barcelona, Barcelona, Spain

Alberto Zangrillo Vita-Salute San Raffaele University, Scientific Institute Ospedale San Raffaele, Milan, Italy

Contents

Foreword V
Frank J. Veith

Preface VII
Germano Melissano, Roberto Chiesa

I – Disease of the Proximal Aorta

1. **Extended use of the FET technique in acute and chronic thoracic aortic disease** 3
Martin Czerny, Tim Berger

2. **Aortic arch aneurysm: hybrid versus total endovascular treatment** 11
Franco Grego, Michele Piazza, Francesco Squizzato, Michele Antonello

3. **Debranch first technique in Frozen Elephant Trunk procedure: the experience of San Raffaele Hospital** 23
Alessandro Castiglioni, Paola Redaelli, Luca Bertoglio, Alessandro Verzini, Marta Bargagna, Ilaria Giambuzzi, Roberto Chiesa, Ottavio Alfieri

4. **Situation specific surgical approach to treat acute Type I dissection complicated by severe malperfusion** 33
Heinz Jakob, Konstantinos Tsagakis

5. **The development of Personalised External Aortic Root Support (PEARS) 2000-2018: a solution for the 21st Century?** 41
Tom Treasure

6. **Ross operation in pediatric age** 51
Alessandro Frigiola, Alessandro Varrica, Mauro Lo Rito

II – Fenestrated and Branched Endografts for Thoraco-abdominal Aortic Disease

7. **Imaging for advanced aortic interventions** 63
Alan B. Lumsden, Ali Irshad

8. **Anatomical and clinical implications secondary to thoracic endograft stiffness** 73
Vincent Riambau, Carla Blanco, Laura Capoccia, Gaspar Mestres, Xavier Yugueros

9. **Fenestrated aortic endografting in female patients. Is there a difference?** 81
Tara M. Mastracci, Debra S.T. Chong

10. Cerebral injury during TEVAR: new causes and new solutions 85
Tilo Kölbel, Vladimir Makaloski, Fiona Rohlffs

11. Do we need an inferior mesenteric artery branch in selected cases? 93
Piotr M. Kasprzak, Kyriakos Oikonomou, Wilma Schierling, Karin Pfister

12. Different strategies of staging the endovascular treatment of thoraco-abdominal aneurysms with fenestrated and branched endografts 99
Luca Bertoglio, Andrea L. Kahlberg, Maria Katsarou, Germano Melissano, Roberto Chiesa

13. Surgeon-modified endografts and off-the-shelf multi branched devices in complex aortic emergencies 113
Nikolaos Tsilimparis, Nikolas Konstantinou, Franziska Heidemann, Tilo Kölbel

14. Fenestrated and branched endograft after previous open infra-renal aortic aneurysm repair 121
Mauro Gargiulo, Enrico Gallitto, Jonathan Sobocinski, Rodolfo Pini, Cecilia Fenelli, Gianluca Faggioli, Chiara Mascoli, Andrea Stella, Stephan Haulon

15. Upper extremity access for complex EVAR/TEVAR 131
Ross Milner, Trissa Babrowski

16. Indications and results of iliac branches in the treatment of complex aortic aneurysm 137
Carlo Pratesi, Fabrizio Masciello, Giovanni Pratesi, Sara Speziali, Aaron Fargion

III – Type B Aortic Dissection

17. Consistency of predictors of aortic events in uncomplicated Type B aortic dissection 151
Santi Trimarchi, Domenico Spinelli, Viviana Grassi, Filippo Benedetto

18. Treatment of chronic dissections with fenestrated and branched endografts: technical aspects 161
Eric L.G. Verhoeven, Pablo Marques de Marino, Athanasios Katsargyris

19. The role of branched devices to treat late complications of Type B aortic dissections 169
Marcelo Ferreira, Diego Ferreira, Rodrigo Cunha, Eduardo Rodrigues, Guilherme Bicalho

20. The STABILISE technique: an evolution of the PETTICOAT for acute and subacute aortic dissection 179
Daniele Mascia, Andrea L. Kahlberg, Luca Bertoglio, Diletta Loschi, Alessandro Grandi, Germano Melissano, Roberto Chiesa

21. Intimal disruption (STABILISE concept) by dilation of thoraco-abdominal aortic bare stents to obtain complete distal sealing in both acute and chronic aortic dissections 187
Jean-Marc Alsac, Elsa Madeleine Faure

22. Thoracic aorta remodeling after endovascular treatment 195
Domenico Palombo, Giovanni Spinella, Valerio Gazzola, Alice Finotello, Simone Mambrini, Michele Conti, Ferdinando Auricchio, Bianca Pane

IV – Abdominal Aortic Aneurysms

23. Endovascular options for management of infrarenal abdominal aortic aneurysm following aortic dissection 205
Ronald L. Dalman, Anahita Dua

24. Preventive mesh augmentation following open aortic repair: current evidence 213
Eike Sebastian Debus, Tilo Kölbel, Sabine H. Wipper, Nikolaos Tsilimparis, Alex Larena-Avellaneda, Holger Diener

25. Open conversion for failed EVAR: increasing need and different strategies 219
Michel S. Makaroun, Abhisekh Mohapatra, Michael J. Singh

26. Should we screen women for abdominal aortic aneurysm? 229
Janet T. Powell and the SWAN collaborators

27. Endovascular repair of ruptured abdominal aortic aneurysms: history, key strategies, adjuncts and present status 233
Frank J. Veith, Neal S. Cayne, Mario L. Lachat

28. Ruptured aneurysm protocol: lessons from a busy aortic center 241
Benjamin W. Starnes

29. Is there a place for open abdominal aortic aneurysm surgery in 2018? 247
Arno von Ristow

30. Endovascular solutions for the treatment of short necks 253
Armando Mansilha, José Oliveira Pinto

31. Optimal treatment to lower blood pressure at the time of pre-operative cardiac work-up may decrease Type II endoleak prevalence after elective EVAR 259
Stefano Michelagnoli, Patrizia Lo Sapio, Emiliano Chisci, Cristina Botteri, Aldo Alberti, Rossella Marcucci

32. Endovascular treatment of juxtarenal aneurysms unfit for fenestrated endograft: the VENT technique 265
Carlo Setacci, Gianmarco de Donato, Francesco Setacci, Edoardo Pasqui, Mariagnese Mele, Claudia Panzano, Domenico Benevento, Giancarlo Palasciano

33. Results of a commercially available unibody stent-graft implantation in patients with TASC D aortoiliac lesions and coexistent abdominal aortic aneurysms 275
Francesco Speziale, Pasqualino Sirignano, Wassim Mansour, Laura Capoccia, Simone Cuozzo

34. Laparoscopic aortic surgery 283
Marc Coggia, Isabelle Javerliat, Raphaël Coscas, Elda Chiara Colacchio, Olivier Goëau-Brissonnière

35. Turning ruptured EVAR into an elective EVAR 291
Isabelle Van Herzeele and Zoran Rancic on behalf of MARZ (Management of Aortic Rupture Zurich) enthusiasts

V – Organ Protection

36. Evoked potentials for neurophysiologic monitoring during aortic surgery 305
Ubaldo Del Carro, Francesca Bianchi, Marco Cursi, Heike Caravati

37. Staging based on intraoperative neuromonitoring findings during thoraco-abdominal aortic aneurysm repair 313
Gustavo S. Oderich, Emanuel R. Tenorio, Jussi M. Kärkkäinen

38. Cerebral protection in aortic arch surgery 327
Roberto Di Bartolomeo, Davide Pacini, Giacomo Murana, Ciro Amodio, Luca Di Marco, Alessandro Leone

39. Effects of renal protection with Custodiol solution during supra-renal and thoraco-abdominal aortic surgery 341
Yamume Tshomba, Angela M.R. Ferrante, Marco Leopardi, Germano Melissano, Roberto Chiesa

VI – Infections

40. Surgery for postoperative infectious aneurysms of the aortic arch and descending thoracic aorta 351
Leo A. Bockeria, Valeriy S. Arakelyan

41. Radical surgical strategies for infected thoracic endografts and esophageal fistula 361
Michael J. Jacobs, Alexander Gombert, Drosos Kotelis

42. Mycotic aortic aneurysms: a paradigm shift in surgical treatment 367
Anders Wanhainen, Karl Sörelius, Kevin Mani

43. Aortoesophageal fistula following open and endovascular thoracic aortic repair: management strategies 373
Andrea L. Kahlberg, Alessandro Grandi, Diletta Loschi, Luca Bertoglio, Germano Melissano, Roberto Chiesa

44. Negative pressure dressing for the prevention of wound complications after aortic surgery 385
Domenico Baccellieri, Alessandro Grandi, Germano Melissano, Roberto Chiesa

VII – Miscellanea

45. Stent graft therapy for Marfan syndrome 397
James H. Black III, Caitlin W. Hicks

46. Aortic repair in patients with Marfan syndrome 403
Joseph S. Coselli, Alice Le Huu, James J. Suffoletta, Mary Bo Kyung Kim

47. Surgery in aortoarteritis 415
Bashi V. Velayudhan, A. Mohammed Idhrees

48. Simultaneous treatment of coral reef atherosclerosis of the visceral aorta and aortoiliac occlusive disease 431
Laurent Chiche, Jean-Michel Davaine, Mahine Kashi, Giulia Rossi

49. New technologies and devices in percutaneous and endovascular endoleak treatments 445
Gianpaolo Carrafiello, Chiara Floridi, Ejona Duka

50. The fate of patients not operated for descending thoracic and thoraco-abdominal aortic aneurysm 453
Patrizio Castelli, Gabriele Piffaretti, Marco Franchin, Stefania Ferraro, Antonino Tarallo, Maria Cristina Cervarolo, Matteo Tozzi

51. Endovascular treatment of juxtarenal aortic occlusion 465
Nicola Mangialardi, Sonia Ronchey, Matteo Orrico, Francesco Aloisi, Vittorio Alberti, Alessio Vona, Fabrizio Nesi, Mario Marino

52. Point-of-care for bleeding management in thoraco-abdominal aortic aneurysm open repair 475
Fabrizio Monaco, Pasquale Nardelli, Gaia Barucco, Cristina Mattioli, Monica De Luca, Alberto Zangrillo

53. Current status of aortic interventions in Egypt 485
Mohamed Hosni Eldessoki

54. Growing a global movement of aortic disease awareness in real life. Turning theory to practice 491
Timo Söderlund

20

The STABILISE technique: an evolution of the PETTICOAT for acute and subacute aortic dissection

Daniele Mascia, Andrea L. Kahlberg, Luca Bertoglio, Diletta Loschi, Alessandro Grandi, Germano Melissano and Roberto Chiesa

20.1 Introduction

Acute and subacute Type B aortic dissection (TBD) is a serious condition that requires treatment in the presence of complications such as impending rupture, rapid dilatation, intractable pain, hypertension, and especially malperfusion of organs or limbs.

Endovascular therapy of complicated acute/subacute TBD has been recently addressed as the *gold standard* of treatment, when anatomically feasible, due to its reduced invasiveness and the lower operative risk when compared to open surgical repair. Traditionally, the principal aim of endovascular approach to TBD is to close the proximal entry tear by deploying a covered stent-graft, therefore redirecting the blood flow in the true lumen (TL), and promoting thrombosis of the false lumen (FL). A positive remodeling of the aorta (FL reduction / obliteration) and, occasionally, dissection healing may be obtained; however, in the majority of cases, some degree of FL perfusion is maintained through additional tears in the thoracoabdominal aorta and iliac arteries. False lumen persistent perfusion may be responsible, in the acute setting, for lasting TL compression and concurrent organs ischemia. In the chronic setting, it may account for an increased risk of aneurysmal degeneration, especially in the abdominal aortic segment, that remains significant over the years; up to 40% of patients will undergo re-interventions or suffer a complication [1, 2].

To help mitigate this problem, bare stents may be deployed in the thoracoabdominal aorta distally to the covered stent-graft, in order to increase TL size, thus treating dynamic malperfusion and to stabilize the intimal lamella. This technique, also known as the "provisional extension to induce complete attachment technique" (PETTICOAT) offers good short- and mid-term results [3-10]; however, some degree of perfusion of the FL is still maintained, and the aorta still has a tendency to grow distally to the stent-graft [5, 6, 11].

Hofferberth and coworkers in 2012 proposed a further modification of this technique, consisting of ballooning the TL inside the stent-graft and the distally deployed bare stents to rupture the lamella and allow full expansion of the stent in a single channeled aorta; it is known as

Daniele Mascia (mascia.daniele@hsr.it)
Scientific Institute Ospedale San Raffaele,
Milan, Italy

G. Melissano - R. Chiesa (eds), Aortic Complexities

the "stent-assisted balloon-induced intimal disruption and relamination in aortic dissection (AD) repair technique" (STABILISE) [12]. It produced excellent results in the authors' experience, especially with regards to the relief of malperfusion, and the reduction of reintervention rate. Despite the reported satisfactory results, the proposed technique did not gain immediate acceptance in the vascular community, mainly because of concerns regarding the potential risk of rupturing the aorta during ballooning.

20.2 The modified "STABILISE" operative technique

The STABILISE technique used at our center (Fig. 20.1) is an evolution of the PETTICOAT technique that was previously reported in detail [7].

It follows most of the concepts initially proposed by Hofferberth et al. [12], with some restrictions in its use, and adds important technical modifications in order to reduce possible risks of aortic rupture. It is mainly based on the following rules:

1) Only patients presenting with an acute or subacute complicated TBD with a proximal suitable non-dissected landing zone in the aortic arch or descending thoracic aorta are considered for this kind of treatment (supra-aortic trunks debranching may be employed to obtain an adequate proximal landing zone (PLZ)).
2) The total aortic diameter (TL + FL) of the abdominal aorta (from supra-celiac to infrarenal level) must not exceed 42 mm.
3) A proximal covered stent-graft is deployed covering the proximal dissection entry tear, using a maximum 10% graft oversizing as compared to the non-dissected PLZ (outer-to-outer diameter).
4) A second (distal) covered stent-graft may be deployed in the descending thoracic aorta if needed, allowing for a generous overlap (at least 5 cm) with the proximal component.

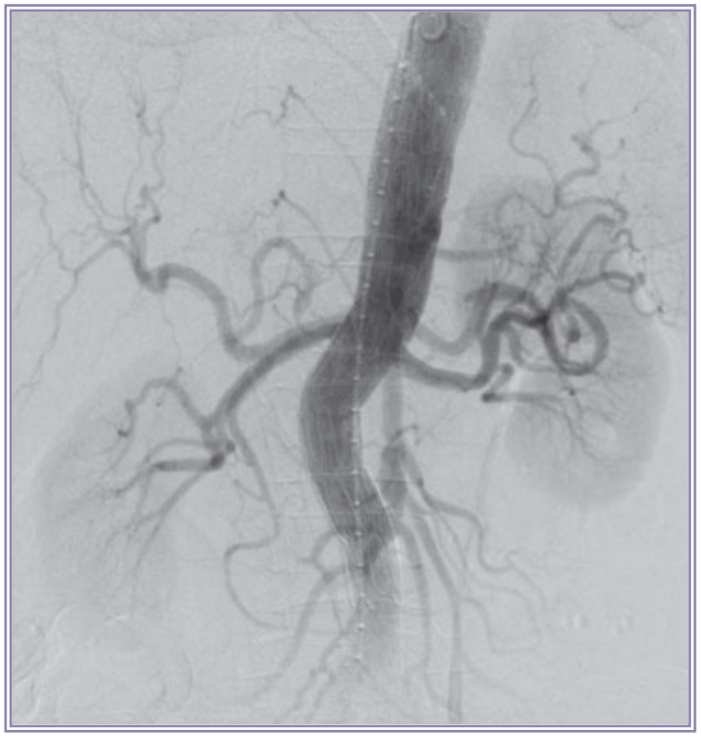

Fig. 20.1 Intraoperative completion angiography depicting patency of all visceral and renal vessels and complete obliteration of the FL.

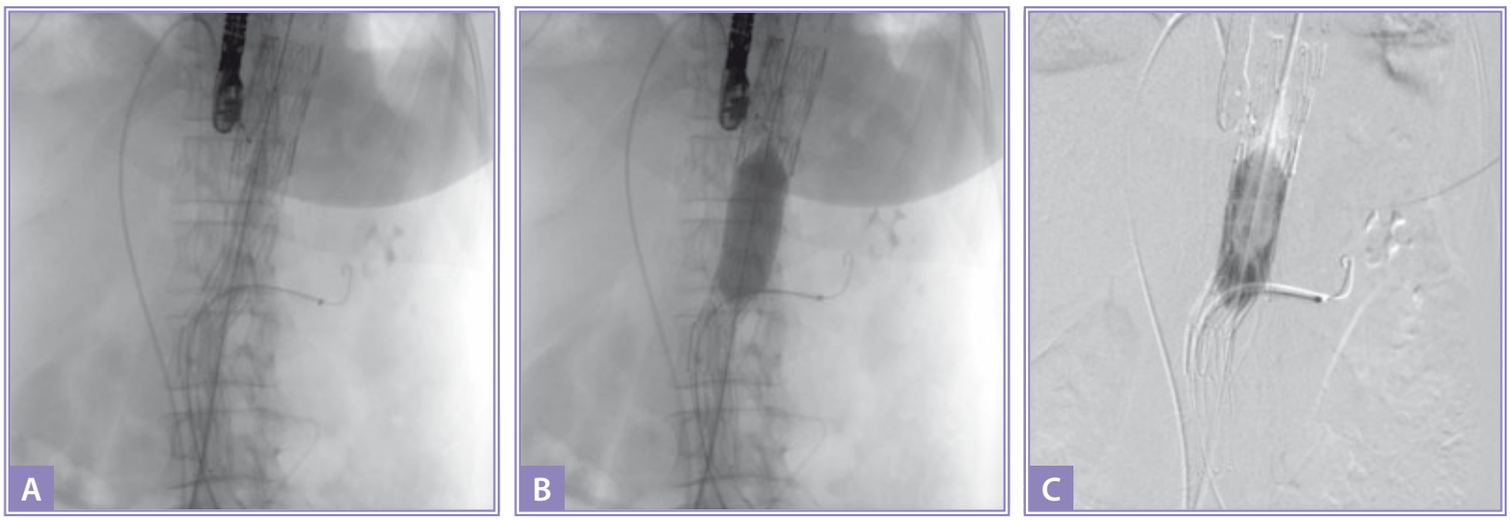

Fig. 20.2 Intraoperative angiography showing the disruption of the FL by inflating a non-compliant balloon in the abdominal aorta (**A**-**B**) with an introducer sheat placed in the left renal artery to avoid its occlusion by the lamella disruption. (**C**) Subtraction angiography shows the movement performed by the introducer sheat during balloon inflation demonstrating its importance in protecting the ostium of the renal artery.

Distal bare-stent configurations are avoided. Distal covered stent-graft diameter is chosen using a maximum 10% graft oversizing as compared to the total aortic diameter (TL + FL) at this level.

5) Distally to the covered stent-grafts, one or more aortic bare stents (aortic dedicated devices) are deployed to cover the entire dissected abdominal aortic segment, with a proximal overlap of at least one stent. Bare stent diameter is chosen to be at least equal to the total aortic diameter (TL + FL) at this level.
6) A 46-mm latex compliant balloon is used to dilate the covered stent-grafts, to rupture the intimal lamella and obtain relamination (i.e., complete FL obliteration) of the descending thoracic segment within the covered stent-graft. In this area, the dilatation of the balloon is constrained within the stent-graft nominal diameter, its fabric protecting the aorta from over-distension.
7) The abdominal aortic bare-stents are then dilated using a non-compliant or semi-compliant balloon, not exceeding the total aortic diameter (TL + FL) at this level, to rupture the intimal lamella and obtain relamination at the abdominal level as well. Balloon-dilatations are constantly followed both radiologically and with transesophageal echocardiography (TEE) where possible (Fig. 20.2). In case of incomplete expansion of the bare stents to the outer aortic wall, ballooning is repeated two more times. After three inflations, no additional maneuvers are performed.
8) In case one or more branches arise from the FL, these are catheterized with a 6 Fr introducer sheath coming from the TL before ballooning; after completion of the STABILISE technique, a bare or covered stent may be deployed to connect the aortic TL with the target vessel (Fig. 20.3).
9) Additional bare or covered stents might be used in the common iliac arteries to obliterate distal tears (Fig. 20.4).

All the procedures are performed under general anesthesia. Cerebrospinal fluid drainage is inserted preoperatively, and the Liquoguard (Möller Medical GmbH, Fulda, Germany) device (which allows monitoring of both the intratecal pressure and the quantity of the fluid drained) was employed with a target intratecal pressure of 8 mmHg or less [13]. If a plug needs to be deployed at the origin of the left subclavian artery in PLZ 2 the left radial artery is catheterized with a 6 Fr introducer sheath. If the LSA is dissected, both TL and FL are plugged.

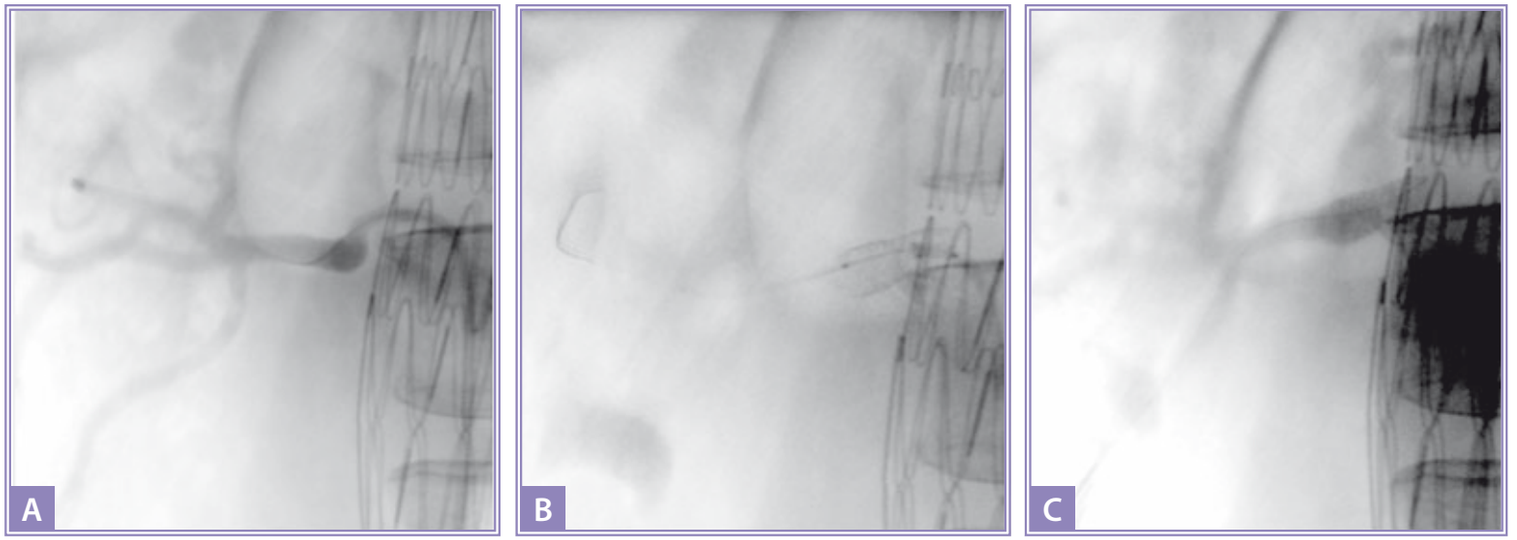

Fig. 20.3 Intraoperative angiography showing (**A**) a stenosis at the origin of the right renal artery after bare metal stent placement in the abdominal aorta, (**B**) stenting of the origin of the left renal artery, and (**C**) completion angiography showing patency of the stented renal artery.

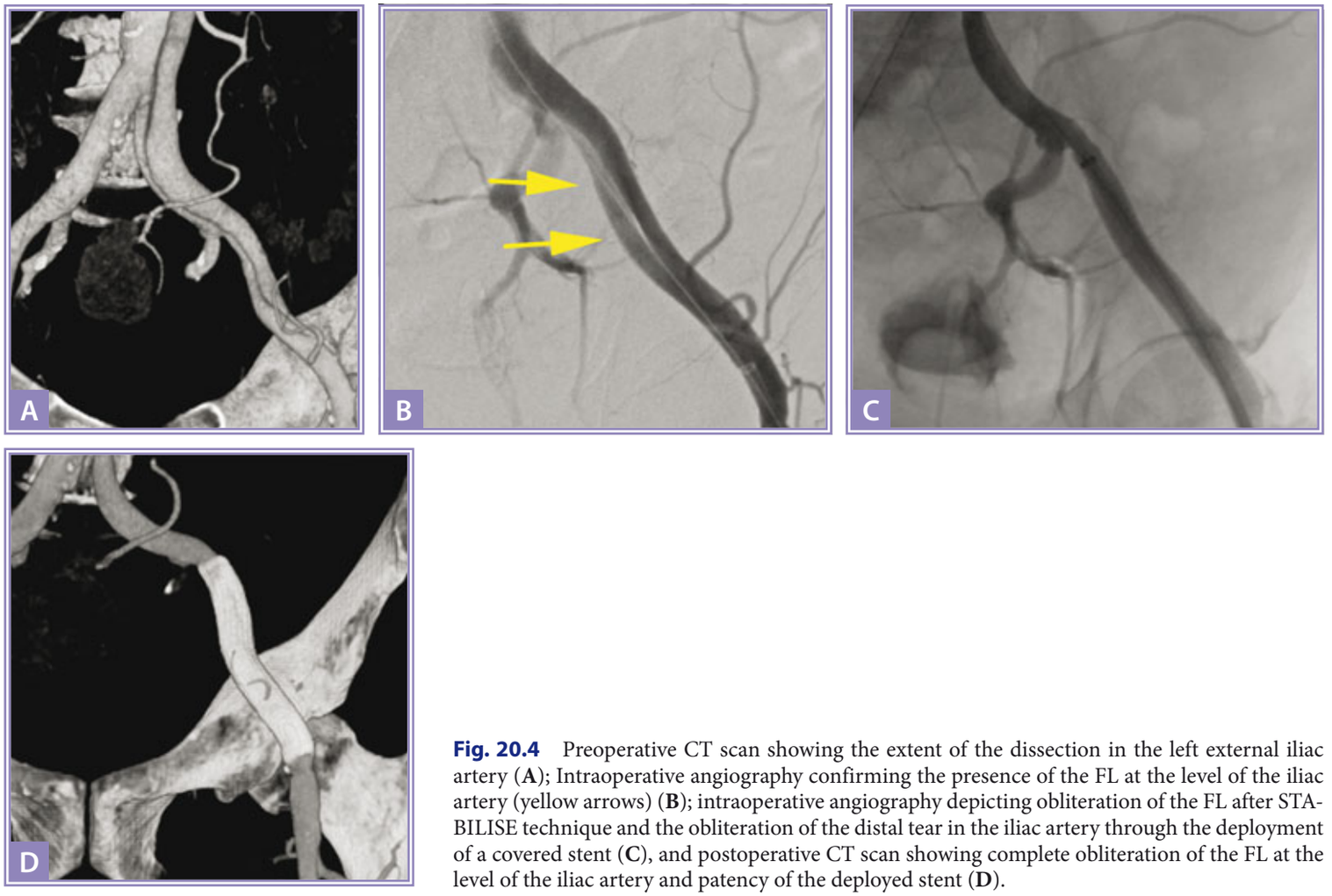

Fig. 20.4 Preoperative CT scan showing the extent of the dissection in the left external iliac artery (**A**); Intraoperative angiography confirming the presence of the FL at the level of the iliac artery (yellow arrows) (**B**); intraoperative angiography depicting obliteration of the FL after STABILISE technique and the obliteration of the distal tear in the iliac artery through the deployment of a covered stent (**C**), and postoperative CT scan showing complete obliteration of the FL at the level of the iliac artery and patency of the deployed stent (**D**).

In most cases, the left common femoral artery is catheterized percutaneously with a 6 Fr 90-cm long introducer sheath for angiographies, and the right common femoral artery was surgically exposed for deployment of the devices. The right femoral vein is also exposed through the groin, and an electrocatheter (Pacer; St. Jude Medical, Minnetonka, MN, USA) is advanced into the right ventricle for rapid cardiac pacing when needed. This routine might be varied in some specific cases according to anatomo-pathological features of the case.

Transesophageal echocardiography is performed in all cases to monitor the cardiac function as well as the positions of the wires at the thoracic level, the deployment of the devices, the ballooning, and the final outcomes. Heparin is administered at an initial dose of 70 U/kg and is supplemented as needed to maintain an activated clotting time (ACT) over 300 seconds.

The TL of the dissected aorta is catheterized with a 5 Fr pigtail catheter that is carefully advanced, with angiographic checks performed during its advancement in the abdomen and with TEE in the thorax. In addition to TEE and angiographic images, in complex cases intravascular ultrasound (IVUS) can be used during the procedure to better understand TL/TF guidewire positioning. Once the tip of the pigtail catheter reached the ascending aorta, a Lunderquist extra-stiff guidewire is inserted and parked adjacent to the aortic valve plane.

Completion angiographies are performed both at the proximal level and at the splanchnic level in order to confirm FL obliteration, and determine patency of all aortic branches. After all the sheaths are removed and the access are closed, patients are immediately awakened to evaluate neurologic integrity.

Protamine sulfate is not routinely administered. Patients are usually monitored in the intensive care unit (ICU), and transferred to the ward after 24 hours if hemodynamically stable and asymptomatic. A CT scan is routinely performed within the first postoperative week prior to discharge. Control CT scans are then performed at 3 months, 6 months, and yearly thereafter.

20.3 San Raffaele experience

Between June 2016 and June 2017, we evaluated 27 patients at our institution for acute and subacute TBD. Ten consecutive patients (all males; mean age 62.6 ± 7.4 y) with acute or subacute complicated TBD, involving the thoracoabdominal aorta who met the anatomic criteria, received treatment with the STABILISE technique. Reasons for treatment included malperfusion (involving the visceral organs in six cases, and the lower limbs in two cases), rapid dilatation in one case, and intractable pain or hypertension in three cases (some patients had more than one presenting symptom). All the patients with complicated TBD received more than one CT scan and were strictly monitored before intervention.

In 6 cases, a supra-aortic trunks rerouting was required (four left carotid-to-subclavian bypass, and two right carotid-to-left carotid-subclavian bypass). A Zenith (Cook Medical, Bloomington, IN, USA) proximal stent-graft (TX2 dissection endovascular graft in 3 cases, or Alpha Thoracic endovascular graft in seven cases) was deployed to land in an area of proximally non-dissected aorta in all cases. A second proximal component (with no distal bare stent) Zenith stent graft (TX2 dissection endovascular graft in four cases, or Alpha Thoracic endovascular graft in seven cases) was then deployed distally with an overlap of at least 5 cm.

Ballooning of the stent-grafts and bare stents immediately obtained complete disruption of the dissecting lamella in 8 cases. In 2 patients after three inflations at the same stent level, only a partial disruption of the abdominal lamella was achieved. Adjunctive endovascular procedures included: 7 cases of stenting of the left renal artery arising preoperatively from the FL, one case of stenting of the superior mesenteric artery arising from the TL with static dis-

section at the origin, and 2 cases of endovascular exclusion of distal secondary re-entry tears with covered stent deployment in the iliac arteries.

On awakening, all patients were neurologically intact and were extubated in the operating room before they were transferred to the ICU for monitoring. Malperfusion immediately resolved in all patients with this initial indication. Posterior thoracic pain was a common complaint (six patients) in the first 24 to 48 hours; it responded well to medication and resolved spontaneously in all cases within discharge.

All patients were monitored for 24 hours in the ICU and then transferred to the vascular surgical ward, where they were followed for a week. One patient experienced paraparesis on postoperative day 5, following an episode of mild hypotension. He fully recovered after reinsertion of cerebrospinal fluid drainage and hemodynamic normalization.

Technical success was obtained in all patients. No deaths were recorded in the hospital or within 30 days postoperatively. No other complications besides the paraparesis were observed in the hospital or within 30 days postoperatively.

The follow-up period ranged from 1 to 24 months (mean 12.3 mo). All patients were alive and clinically asymptomatic during this follow-up period. The thoracic FL was either completely excluded or had entirely disappeared in all, but one case of mild reperfusion sustained by intercostal arteries (Fig. 20.5).

The total diameter of the thoracic aorta, including TL and FL, decreased in all patients who preoperatively presented a dilatation of the FL, and remained stable in all cases of non-dilated thoracic aorta. In the abdominal aorta, the TL was widely patent in all cases with no signs of malperfusion. Some degree of perfusion was observed between the bare stents and the external

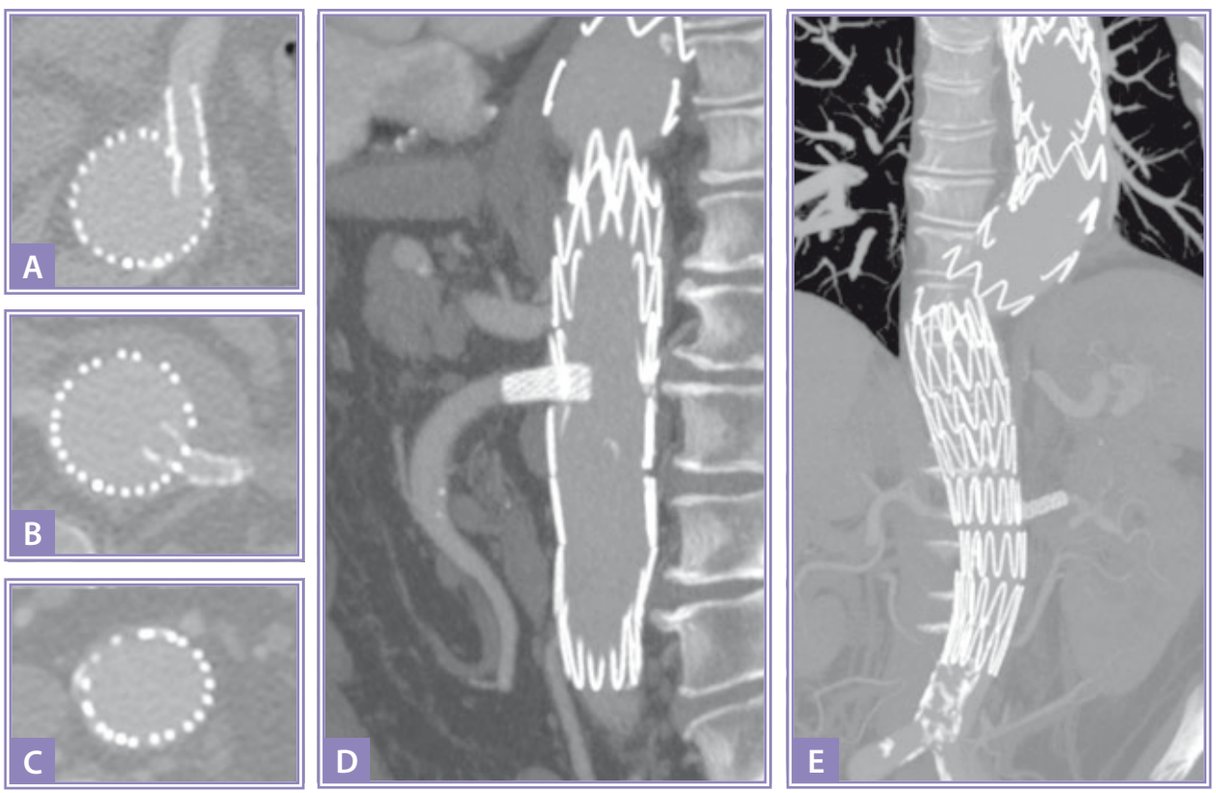

Fig. 20.5 A follow-up CT scan showing (**A**) obliteration of the FL and patency of a stent placed in the superior mesenteric artery, (**B**) obliteration of the FL and patency of a stent placed in the left renal artery, (**C**) complete obliteration of the FL at the level of the abdominal aorta, (**D**) 3D reconstruction showing the obliteration of the FL and patency of a stent placed in the superior mesenteric artery, and (**E**) 3D reconstruction showing the obliteration of the FL and patency of a stent placed in the left renal artery.

aortic wall in 2 patients with only partial intraoperative lamella disruption. No further maneuvers were performed so far in these 2 cases. The diameter of the abdominal aorta remained stable in all cases, and no abdominal aortic enlargement was documented at current follow-up.

After 6 months of follow-up, one patient presented with asymptomatic right renal stenosis, successfully treated with a 7-mm balloon-expandable stent. No other patency issues were observed in splanchnic and renal vessels.

20.4 The "STABILISE registry"

While several authors reported favorable results with this technique, ballooning of the TL after PETTICOAT lies outside the IFU of the bare stents; therefore, more robust data are needed in order to gain consensus and the IFU modified accordingly. In October 2018, a protocol study for the first physician-initiated, international, multi-center, non-randomized, observational registry of patients with acute/subacute Type B AD treated by means of the STABILISE technique was approved by the San Raffaele Scientific Institute Ethical Committee. With this study, we aim to gather – for the first time – homogeneous data coming from high-volume international aortic centers performing the STABILISE technique, in order to gain a hard scientific basis to evaluate its results, and compare them to the currently used alternative strategies. The primary end-point of this study is considered the 30-day clinical success, defined as the successful procedure execution, visceral/iliac vessel patency and no major adverse event (all-cause mortality, bowel ischemia, myocardial infarction, paraplegia, respiratory failure, stroke and renal insufficiency). The secondary end-points are mortality at 6 and 12 months, persistent false lumen perfusion, aortic enlargement requiring open conversion, aortic related reinterventions, visceral vessels patency, iliac arteries patency, visceral vessels reintervention for in-stent restenosis/occlusion and spinal cord ischemia.

The study is a prospective and retrospective observational registry, including all patients with acute/subacute (up to 90 days from the onset) Type B AD treated by means of the stent-assisted balloon-induced intimal disruption and relamination in AD repair (STABILISE) technique. The study is designed to enroll up to 200 patients. The follow-up will continue up to 2 years. All patients will be followed according to normal clinical practice and a thoraco-abdominal CT scan will be provided within 1 month, within 6 months, after 1 year and after 2 years from the procedure.

All patients will have to abide to the following inclusion criteria:

- patients with ≥ 18 years of age, presenting with an acute/subacute (up to 90 days from the onset of dissection) Type B dissection with a proximal suitable non-dissected landing zone in the aortic arch or descending thoracic aorta (supra-aortic trunks debranching may be employed to obtain an adequate proximal landing zone);
- total aortic diameter of the abdominal aorta (from supra-celiac to infra-renal level) must not exceed 42 mm;
- patients treated with the STABILISE technique according to the following treatment protocol;
- patients able to sign specific informed consent for the study.

And the following exclusion criteria:

- patients with chronic Type B AD (after 90 days from the onset of dissection);
- patients with acute Type B AD not treated according to the following treatment protocol;
- unwilling or unable to comply with the follow-up schedule;
- inability or refusal to give informed consent;
- simultaneously participating in another investigative device or drug study;
- frank rupture;
- systemic infection (eg, sepsis);

- allergy to stainless steel, polyester, solder (tin, silver), polypropylene, nitinol, or gold;
- untreatable reaction to contrast, which, in the opinion of the investigator, cannot be adequately premedicated;
- surgical or endovascular AAA repair within 30 days before or after dissection repair;
- interventional and/or open surgical procedures (unrelated to dissection) within 30 days before or after dissection repair.

This physician-initiated voluntary registry is not supported by the industries and strongly requires the collaboration of all colleagues to enroll patients and obtain results in a reasonable time. It is open to all centers performing this technique within the protocol limits. Interested centers are cordially invited to contact the authors to join the registry (stabiliseregistry@gmail.com).

20.5 Conclusions

The PETTICOAT concept has been proved to be a valuable adjunct in cases of persistent malperfusion after an initial TEVAR for TBD; however, it failed to be effective to promote remodeling of the distal thoracoabdominal aorta. The STABILISE concept has been proposed to create a single aortic channel and, therefore, effectively treat dynamic malperfusion, avoiding reinterventions and enhancing aortic remodeling and healing. Further studies are needed to ascertain the safety of this technique in a larger group of patients and to evaluate the behavior of the post-dissected aorta with time.

REFERENCES

1) Fattori R, Montgomery D, Lovato L, et al. Survival after endovascular therapy in patients with type B aortic dissection: a report from the International Registry of Acute Aortic Dissection (IRAD). JACC Cardiovasc Interv 2013; 6: 876-82.
2) Faure EM, Canaud L, Agostini C, et al. Reintervention after thoracic endovascular aortic repair of complicated aortic dissection. J Vasc Surg 2014; 59: 327-33.
3) Ito N, Tsunoda T, Nakamura M, et al. Percutaneous bare Z-stent implantation as an alternative to surgery for acute aortic dissection with visceral ischemia. Catheter Cardiovasc Interv 2003; 58: 95-100.
4) Mossop PJ, McLachlan CS, Amukotuwa SA, Nixon IK. Staged endovascular treatment for complicated type B aortic dissection. Nat Clin Pract Cardiovasc Med 2005; 2: 316-21.
5) Nienaber CA, Kische S, Zeller T, et al. Provisional extension to induce complete attachment after stent-graft placement in type B aortic dissection: the PETTICOAT concept. J Endovasc Ther 2006; 13: 738-46.
6) Hofferberth SC, Newcomb AE, Yii MY, et al. Combined proximal stent grafting plus distal bare metal stenting for management of aortic dissection: Superior to standard endovascular repair? J Thorac Cardiovasc Surg 2012; 144: 956-62.
7) Melissano G, Bertoglio L, Rinaldi E, et al. Volume changes in aortic true and false lumen after the "PETTICOAT" procedure for type B aortic dissection. J Vasc Surg 2012; 55: 641-51.
8) Lombardi JV, Cambria RP, Nienaber CA, et al; STABLE investigators. Aortic remodeling after endovascular treatment of complicated type B aortic dissection with the use of a composite device design. J Vasc Surg 2014; 59: 1544-54.
9) Alsac JM, Girault A, El Batti S, et al. Experience of the Zenith Dissection Endovascular System in the emergency setting of malperfusion in acute type B dissections. J Vasc Surg 2014; 59: 645-50.
10) Kische S, D'Ancona G, Belu IC, et al. Perioperative and mid-term results of endovascular management of complicated type B aortic dissection using a proximal thoracic endoprosthesis and selective distal bare stenting. Eur J Cardiothorac Surg 2015; 48: e77-84.
11) Sobocinski J, Lombardi JV, Dias NV, et al. Volume analysis of true and false lumens in acute complicated type B aortic dissections after thoracic endovascular aortic repair with stent grafts alone or with a composite device design. J Vasc Surg 2016; 63: 1216-24.
12) Hofferberth SC, Boston RC, McLachlan CS, Mossop PJ. Stent-assisted balloon-induced intimal disruption and relamination in aortic dissection repair: the STABILISE concept. J Thorac Cardiovasc Surg 2014; 147: 1240-5.
13) Tshomba Y, Leopardi M, Mascia D, et al. Automated pressure-controlled cerebrospinal fluid drainage during open thoracoabdominal aortic aneurysm repair. J Vasc Surg 2017; 66(1): 37-4.

37

Staging based on intraoperative neuromonitoring findings during thoraco-abdominal aortic aneurysm repair

Gustavo S. Oderich, Emanuel R. Tenorio and Jussi M. Kärkkäinen

37.1 Introduction

Neurological injury is a major limitation of aortic surgery, whether it involves the spinal cord or brain. Endovascular treatment of thoraco-abdominal aortic aneurysms (TAAAs) has greatly evolved in the last decade and currently applied to challenging anatomy such as chronic post-dissection aneurysms and in patients with prior aortic repair. New developments in device design, techniques of implantation, adjunctive measures, and better perioperative care have resulted in substantial improvements in morbidity and mortality [1]. Contemporary reports by experienced operators indicate that 30-day mortality ranges from 5% to 9 %, which compares favorably to the largest open surgical experiences in centers with over thousands of repairs [1-5]. Nonetheless, spinal cord injury (SCI) continues to be the most devastating complication for the patient, family and surgeon, occurring in up to 35% of patients in some series [1].

Permanent paraplegia has a devastating impact on patient's quality of life and long-term prognosis, with a higher complication and mortality rate, longer intensive care and hospital stay [6]. Although the etiology of SCI is multifactorial, arterial embolization and hemodynamic compromise from coverage of large aortic segments, insufficient collaterals, and systemic hypotension are the main predisposing factors during endovascular TAAA repair [2, 7-9]. The spinal cord collateral system includes the carotid, subclavian, vertebral, intercostal, lumbar, hypogastric, and deep femoral arteries [2, 5, 10]. As such, pre-operative, intra-operative, and post-operative strategies can be applied to maximize spinal cord perfusion and decrease risk of SCI.

37.2 Risk stratification

The risk of spinal cord injury is variable depending on a number of predisposing risk factors (Table 37.1). Extent of aortic coverage is the most important predictor with the highest rates

Gustavo S. Oderich (Oderich.Gustavo@mayo.edu)
Mayo Clinic, Rochester, MN, USA

G. Melissano - R. Chiesa (eds), Aortic Complexities

Table 37.1 Risk factors associated with permanent SCI in patients treated with thoracic or fenestrated-branched stent grafts

Study	N.	Permanent SCI	Risk factors	Odds ratio
Feezor *et al.* [11]	326	4%	Age (every 10 years)	1.6 (1.1-2.2, P = 0.006)
			Thoracic aortic coverage (every 20 mm)	1.3 (1.1-1.5; P = 0.0004)
			Uncovered distance from celiac artery to distal margin of graft (every 20 mm)	0.61 (0.42-0.87; P = 0.006)
Drinkwater *et al.* [34]	235	9%	Aortic coverage (every 10%)	1.78 (1.18-2.71, P = 0.007)
Hanna *et al.* [18]	381	7.4%	Age in years	1.05 (1.01-1.09, P = 0.02)
			Number of stents implanted	2.0 (1.4-2.9, P = 0.0002)
			Postoperative hypotension[a]	3.9 (1.7-9.3, P = 0.002)
Scali *et al.* [14]	741	5.1%	Aortic coverage (every 50 mm)	1.3 (1.1-1.6, P = 0.002)
			Hypertension[b]	6.4 (2.6-18, P < 0.0001)
Bisdas *et al.* [12]	142	11%	Thoracic aortic coverage (every 1%)	1.03 (1.01-1.05; P = 0.001)
Dias *et al.* [13]	71	22.5%	Crawford extent II	4.497 (1.331-15.195, P < 0.05)
			Higher contrast volume	3.736 (1.054-13.242, P = 0.041)
Katsargyris *et al.* [19]	218	4%	>300 minutes procedure duration	7.4 (2.6-21.1, P < 0.001)
			Peripheral arterial disease	6.6 (2-21.9, P = 0.002)
			Baseline renal insufficiency[c]	4.1 (2-21.9, P = 0.04)
Sobel *et* al. [26]	116	7.7%	Fluoroscopy time >190 minutes	3.6 (1.0-12.7, P = 0.04)
			Baseline renal insufficiency[c]	4.2 (1.2-14.6, P = 0.03)
			Sustained postoperative hypotension[d]	2.89 (1.07-7.7, P = 0.04)

[a] Defined as any systolic blood pressure <100 mmHg within 72 postoperatively; [b] defined as chart history and/or chronic (>30 days) medication; [c] defined by glomerular filtration rate <30 mL/min; [d] defined as systolic blood pressure <90 mmHg for >15 minutes in operation room.

observed for Extent II (10-20%) and I TAAAs and the lowest rates for Extent IV TAAAs (1-5%) [11-13]. Among patients with complex abdominal aneurysms requiring fenestrated grafts, the risk of spinal cord injury is relatively low; however, this risk is slightly higher among patients who need aortic coverage ≥5 cm above the celiac axis [11, 14]. Presence of occluded collateral network (hypogastric and/or vertebral artery) is associated with higher rate of immediate paraplegia and lack of improvement in motor function [2]. Other determinants include prior aortic replacement, chronic kidney disease, chronic obstructive pulmonary disease and age [15-19]. In general, our approach has been to routinely apply a protocol for spinal cord injury prevention in all patients undergoing complex endovascular aortic repair with ≥5 cm (or two sealing stents) above the celiac axis.

37.3 Mechanisms of injury

Spinal cord ischemia often result from multiple superimposed insults, which may occur in combination or as isolated events [2, 5]. Although many reports have identified risk factors for spinal cord injury, the specific causes have not been well described. Some of the recognized factors are hemodynamic deterioration leading to spinal cord infarction secondary to loss of intercostal arteries coupled with insufficient collateral network; reperfusion injury and spinal cord edema; and microembolization from catheter or device manipulations. Traditionally, embolization has been considered a relatively uncommon cause. However, since the widespread use of adjunctive measures to improve spinal cord perfusion during open aortic repair (e.g., distal perfusion, intercostal reimplantation, Mechanisms of Injury hypothermia, CFS drainage), the most common etiology of spinal cord ischemia may have shifted from hemo-

dynamic deterioration to micro-emboli. Tanaka and associates reported the results of MRI to evaluate mechanisms of spinal cord injury after aortic repair. In that study, diffuse infarction around the *Artery Radicularis Magna*, which is consistent with hemodynamic deterioration, was observed in only 20% of patients with injury. The remaining 80% of patients had scattered or focal MRI lesions corresponding to infarction from microembolization [7].

Prolonged lower extremity ischemia during complex endovascular procedures has been associated with more complications and increased risk of spinal cord injury [5]. The introduction of large sheaths, which are needed for delivery of the aortic and target vessel stents, occlude the inflow to the lower extremity and pelvis by covering the origins of the profunda femoris and internal iliac arteries. The surgeon needs to be attentive to lower extremity ischemia and its deleterious consequences including acidosis, pelvic and spinal ischemia and lower extremity compartment syndrome. Ideally, lower extremity perfusion should as soon as possible.

Recent experimental and clinical studies have shown that staged segmental arterial coverage of the aorta allows rapid recruitment of the spinal collateral networks, decreasing mortality and SCI during endovascular TAAA repair. Bischoff and associates reported no spinal cord injury in pigs randomized to staged coverage compared to 50% rate of paraplegia in pigs treated in a single stage [20]. Subsequently, the Cleveland Clinic group reported that internal iliac artery occlusion was an independent predictor of immediate spinal cord injury and lack of recovery, while staged thoraco-abdominal aortic repair reduced early mortality and rates of paraplegia [2, 10]. The benefits of staged repair for thoraco-abdominal aneurysm may go beyond the optimization of collaterals. It is possible that a faster, simpler, second-stage procedure with earlier restoration of pelvic and lower extremity blood flow is associated with improved spinal cord perfusion, or that prior stent graft coverage of the proximal thoracic aorta results in lower rates of micro-embolization during the second stage procedure or a less pronounced systemic inflammatory response during the second-stage procedure. Although further clinical experiences are needed to confirm these results, current evidence suggests that a staged repair with segmental coverage of the aorta can allow rapid recruitment of the spinal collateral networks, decreasing mortality and SCI during endovascular TAAA repair [10, 21, 22].

37.4 Spinal cord injury prevention protocol

A standardized protocol to reduce risk of spinal cord injury is applied in all patients undergoing endovascular TAAA repair. Procedures are performed by a dedicated endovascular team under total intravenous general endotracheal anesthesia using fixed imaging in a hybrid endovascular room. The anesthetic management consists of propofol and fentanyl or sufentanil infusion at the discretion of the anesthesiologist with avoidance of non-depolarizing muscle relaxants. Succinylcholine is used as the muscle relaxant of choice for induction of anesthesia.

37.4.1 Blood pressure management

Calcium channel blockers and angiotensin-inhibitors are discontinued a week prior to the operation and up to 4-6 weeks after the procedure unless systolic blood pressures are >160 mmHg. The goal mean arterial pressure is targeted at ≥80 mmHg intra-operatively and for the first 72 h after the operation. Blood pressure goals are titrated according to intra-operative neuromonitoring or post-operative examination. If there are changes in neuromonitoring detected during the operation or neurological changes observed on post-operative physical examination, MAP goals are incrementally raised up to 100 mmHg. In addition, transfusion of blood products is indicated in the first 48 h after the procedure to keep a target hemoglobin ≥10 mg/dL and normal coagulation profile prior to removal of the spinal drain.

37.4.2 Cerebrospinal fluid drainage

Routine prophylactic CSF drainage was used in all patients with ≥ 5 cm stent-graft coverage (or 2 sealing stents) above the celiac axis, which was the minimum extent of coverage used for all type IV TAAAs. This has been recently modified to patients with Extent I to III TAAAs and we no longer use routine drainage for Extent IV TAAAs due to low risk of spinal cord injury coupled with the potential risk of severe hemorrhagic complications from drain placement. Spinal fluid pressure was set in a closed, pressure-controlled system at a baseline of 10 mmHg. The spinal drain was opened for 15 minutes every hour with a maximum drainage of 20 mL per hour, after which the drain was clamped for the remainder of the hour. If there were changes in neuromonitoring or the neurological examination, CSF pressure was decreased to 5 or 0 mmHg. CSF pressure was raised to 10 mmHg once neuromonitoring or the neurological examination improved. Spinal fluid drainage is continued for 48 to 72 hours in patients with Extent I–III TAAAs. The CSF drain was removed in patients who had stable hemodynamics and neurological examination after a 6-hour clamping trial.

37.4.3 Neuromonitoring

Neuromonitoring has been extensively used during open surgical repair to help identify ischemia in the anterior and lateral spinal columns, which are sensitive to decreases in oxygen supply [23, 24]. During open TAAA repair, this technique has helped guide selective intercostal artery reimplantation and intra-operative maneuvers designed to improve spinal cord perfusion. Because signal changes can result from dysfunction in any of the recorded pathways, including the brain, peripheral nerves, receptors or muscles, as well as technical issues, interpretation requires correlation with the clinical scenario. During endovascular procedures, early detection of these changes may ultimately help identify patients at increased risk of spinal cord injury and in whom hemodynamic deterioration may be prevented or reversed by use of sequential maneuvers designed to optimize spinal cord perfusion pressure (SCPP) by augmentation of mean arterial pressure (MAP) and reduction of cerebrospinal fluid (CSF) pressure (SCPP = MAP – CSF pressure). These maneuvers raise traditional targets currently applied to endovascular procedures (MAP of 80 mmHg; CSF pressure of 10 mmHg, or maintaining a SCPP > 70 mmHg) and include early restoration of flow to the pelvis and lower extremities. In contrast to open aortic procedures, endovascular procedures do not allow for direct incorporation of intercostal arteries.

Neuromonitoring with continuous motor-evoked potential (MEP) and somatosensory-evoked potential (SSEP) has been widely applied during open TAAA repair to guide reimplantation of intercostal arteries and to optimize distal aortic perfusion. In contrast to open repair, endovascular incorporation of intercostal arteries is not feasible. Nonetheless, immediate recognition of ischemia using neuromonitoring may allow introduction of maneuvers to optimize spinal cord and lower extremity (LE) perfusion and decrease the rate of SCI. Total intravenous anesthesia is used to allow intra-operative neurophysiological monitoring of MEP and SSEPs. A ≥ 75% reduction from baseline-evoked potential amplitude is considered to be significant (Fig. 37.1). Cadwell Elite machines and Cascade software is used to collect data (Cadwell Laboratories, Kennewick, WA) by electromyography (EMG) technologists. Electrical charges sent to the motor cortex through C3, C4 scalp electrodes evoke motor-evoked potentials (MEP) through the motor pathway (Fig. 37.2) which are recorded down the cord over multiple muscles in the upper and lower extremities for at least every 10-15 min. An upper extremity muscle (Extensor Digitorum Communis) is recorded to help differentiate neurogenic impairment such as spinal and lower limb ischemia from non-specific changes.

Bilateral lower extremity muscles are recorded using subdermal EEG electrodes placed in

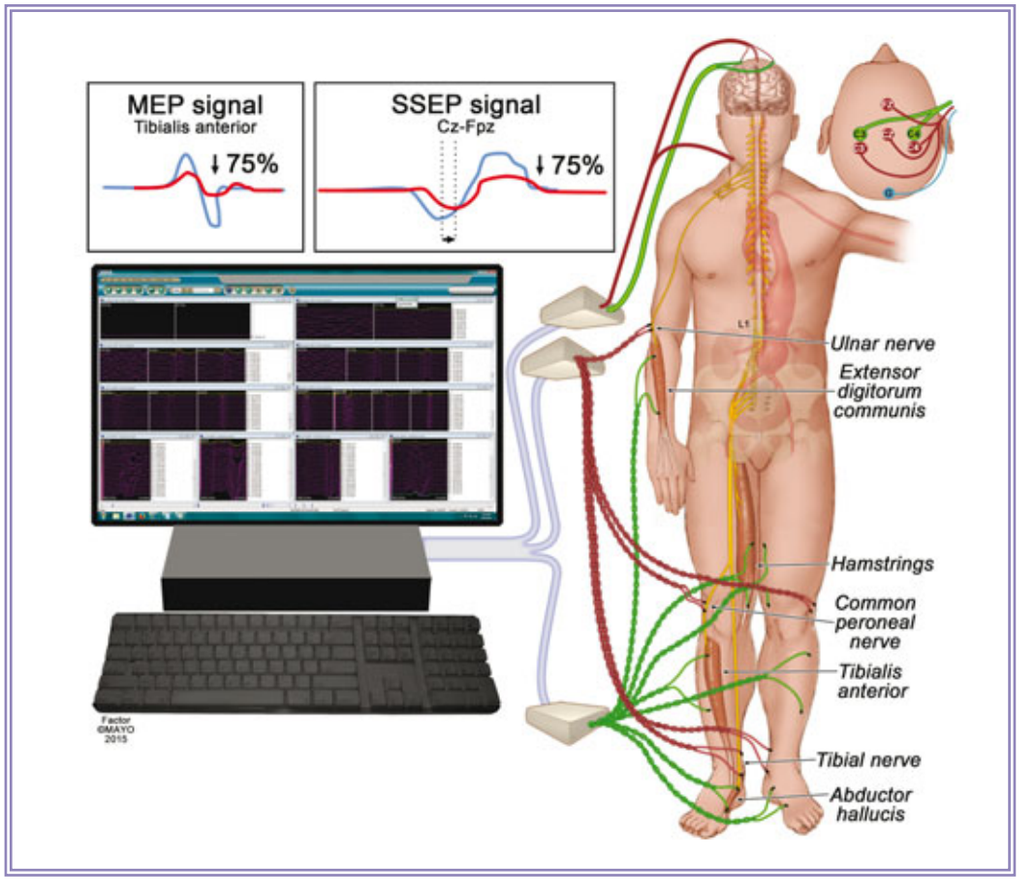

Fig. 37.1 Illustration depicting placement of electrodes for monitoring of motor-evoked (MEP) and somatosensory-evoked potentials (SSEP) during complex endovascular aortic repair. A significant change is defined by greater than 75% decline in amplitude in MEP or SSEPs. By permission of Mayo Foundation for Medical Education and Research. All rights reserved.

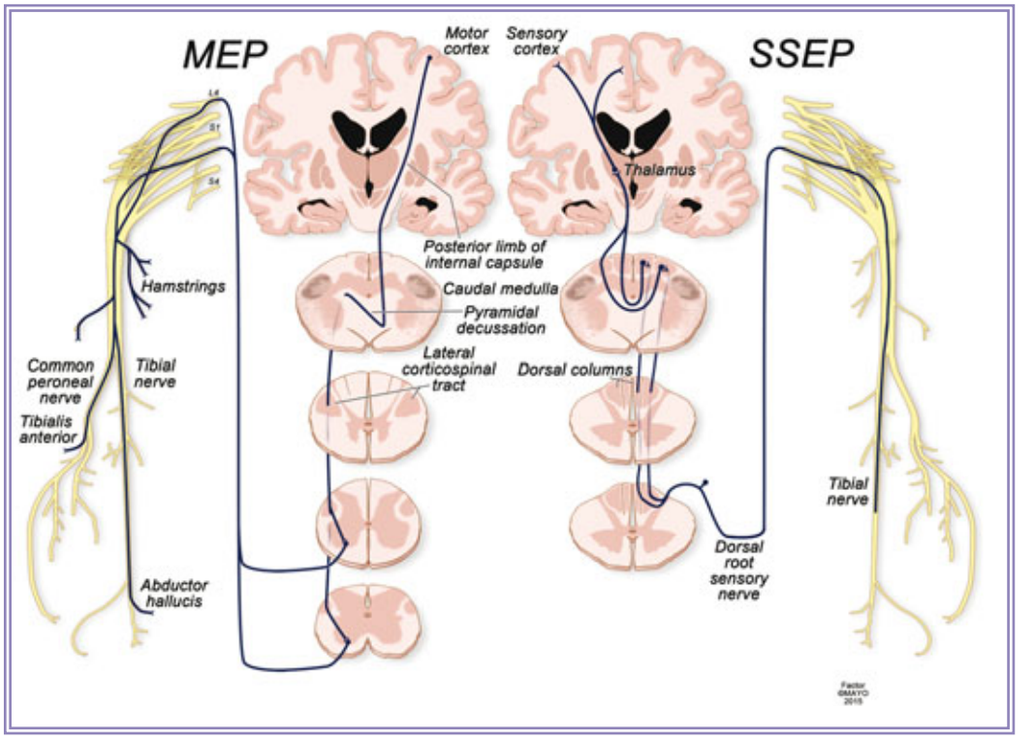

Fig. 37.2 Illustration depicting of motor-evoked (MEP) and somatosensory-evoked potential (SSEP) pathways. By permission of Mayo Foundation for Medical Education and Research. All rights reserved.

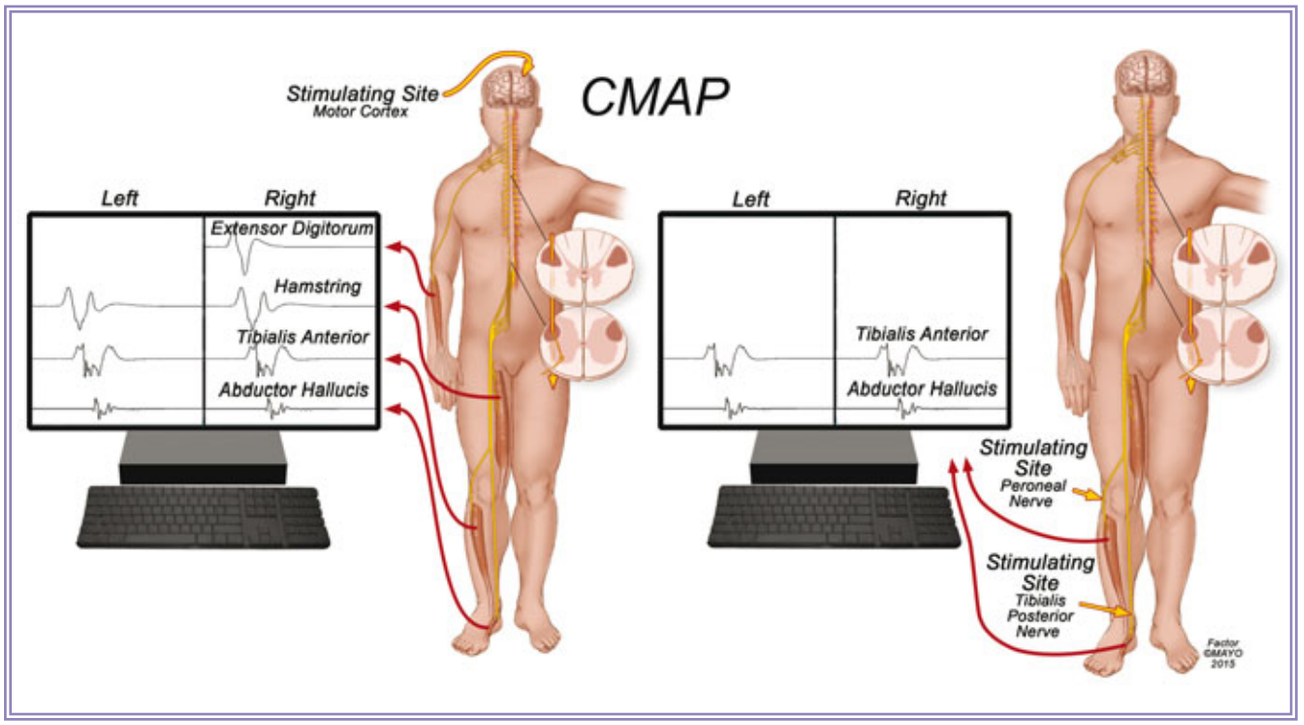

Fig. 37.3 Compound muscle action potentials help differentiate peripheral from spinal cord ischemia. By permission of Mayo Foundation for Medical Education and Research. All rights reserved.

the hamstring, tibialis anterior, and abductor hallucis muscles. In addition, somatosensory evoked potentials (SSEPs) consisting of electrical stimulus generated at the ulnar or tibial nerve that travels from the distal extremity were recorded over the neck and scalp. In patients where the lower extremity tibial SSEP are not present at the ankle, subdermal EEG electrodes can be positioned behind the knee. In addition, a compound muscle action potential (CMAP) is performed by stimulating the peroneal nerve and posterior tibialis nerve (Fig. 37.3). The CMAP is important to help differentiate peripheral nerve and spinal cord ischemia.

37.4.4 Early pelvic and lower limb reperfusion

Large trans-femoral sheaths are associated with limb and pelvic ischemia, which may aggravate spinal cord ischemia because of compromised collateral networks. Increasing evidence indicates that minimizing lower limb and pelvic ischemia may improve outcomes of complex endovascular repair including lower risk of paraplegia [5, 12, 19, 25, 26]. This can be done by several adjunctive measures. The superficial femoral artery may be accessed using a smaller sheath, which can be connected into the larger trans-femoral sheath creating a shunt. However, this technique can be cumbersome and also does not address the occlusion of the internal iliac and profunda femoris arteries, which are the main collateral networks to the spine. Our preference has been to use a temporary iliac artery conduit in patients with small iliac arteries, severe occlusive disease of the aorto-iliac arteries or when technical challenge with prolonged (>2 h) ischemia time is anticipated [25]. A femoral conduit anastomosed end-to-side to the common femoral artery to allow restoration of lower extremity flow. The use of femoral conduit minimizes lower extremity ischemia during visceral branch stenting by allowing the aortic device sheath to be retracted into the conduit (Fig. 37.4). More frequently, we prefer to use total percutaneous femoral approach whenever the common femoral arteries are not calcified

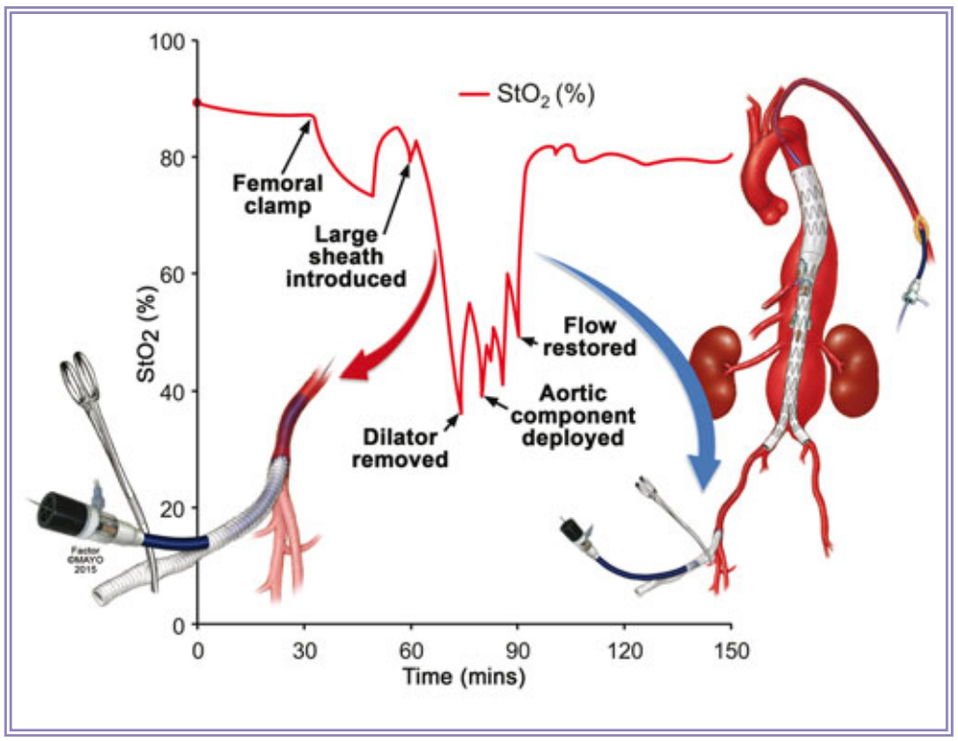

Fig. 37.4 Temporary femoral conduits anastomosed end-to-side to the common femoral artery were used selectively as an adjunct to optimize lower extremity (LE) flow during difficult cases. This graph illustrates the decline in LE trans-cutaneous oxygen saturation (StO_2) with placement of large diameter sheaths in the ilio-femoral arteries. After the dilator is retracted to the femoral conduit, StO_2 returns to baseline values. In this patient there were no changes observed in neuromonitoring, despite the complex anticipated anatomy. By permission of Mayo Foundation for Medical Education and Research. All rights reserved.

and a straightforward case is anticipated. In these cases, the aortic device, renal fenestrated-branches, bifurcated device, and iliac limbs are performed using the femoral approach first. Flow is restored into both lower extremities (<2 h) and the procedure is completed via the left arm by placement of the superior mesenteric and celiac artery stents.

37.4.5 Staging

Staged endovascular approach is used in all patients with extent type I or II TAAAs. Three techniques for staging have so far been implemented and tested, with good results: 1- two-steps aortic coverage; 2-temporary aneurysm sac perfusion (TASP); and 3-minimally-invasive segmental artery coil embolization (MISACE) [10, 27-31]. Our preference is to proceed with coverage of the proximal thoracic aorta from the landing zone to just above the celiac axis, leaving a distal Ib endoleak with completion repair in 6-8 weeks or earlier in patients with rapid expansion of very large aneurysms who are suitable candidates for off-the-shelf devices (Fig. 37.5). Temporary sac perfusion branches can be designed into the main aortic stent, which are left patent in the initial procedure with plan of subsequent occlusion. In these cases, the perfusion branch (es) is occluded in a few days using an amplatzer plug, which can be deployed under local anesthesia (Fig. 37.6). The patient is kept heparinized and observed for 2-4 h with the plug still connected to the delivery system. If there are no changes on examination, the plug is disconnected and heparinization is reversed. Alternatively, the repair can be left incomplete by not placing the contra-lateral iliac limb extension or one of the bridging stents (e.g., celiac stent). Limitations of perfusion branches are the potential risks of increased sac pressure due to poor outflow via small segmental arteries and disseminated intra-vascular coagulopathy from large endoleak into a blind sac. Over time, our technique has evolved and now utilizes sequential aortic coverage by first placing thoracic stent-grafts starting distal to the left subclavian artery and covering up to the celiac artery.

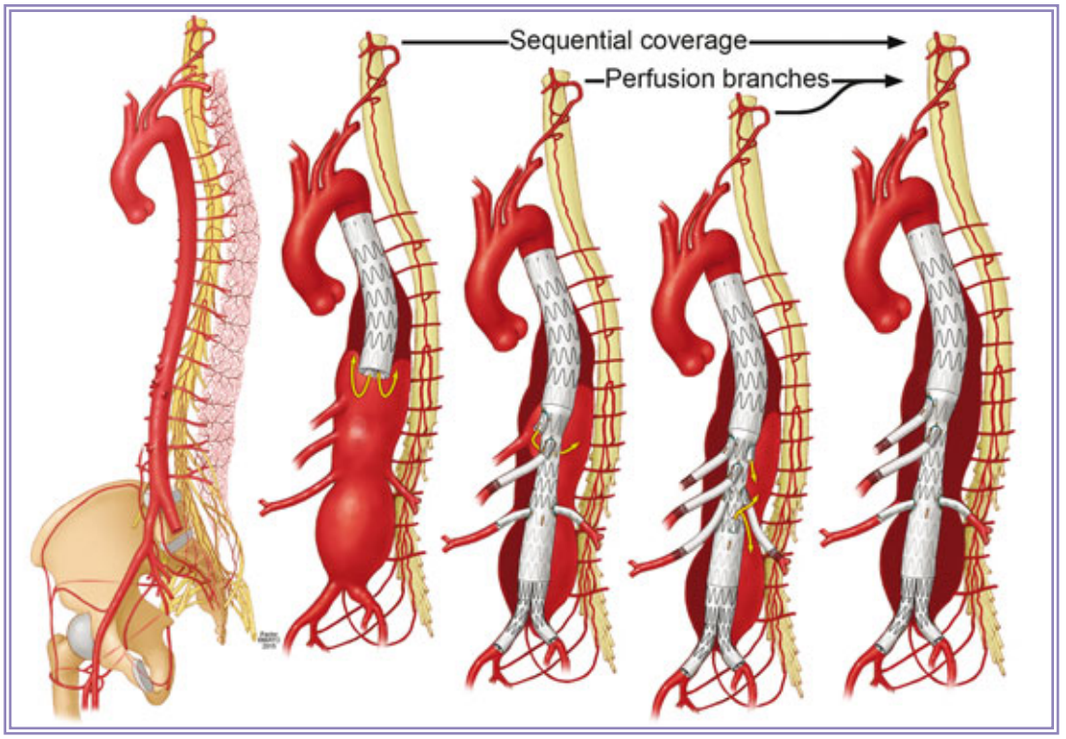

Fig. 37.5 Optimization of the extensive spine collateral network including vertebral, intercostal, lumbar, and hypogastric arteries forms the basis for staged endovascular repair of complex aneurysms. Strategies include coverage of the proximal thoracic aorta up to the celiac axis, followed by visceral branch stenting in a second stage. Alternatively, the sac can be perfused via perfusion branches or unstented celiac axis or contra-lateral iliac limb. By permission of Mayo Foundation for Medical Education and Research. All rights reserved.

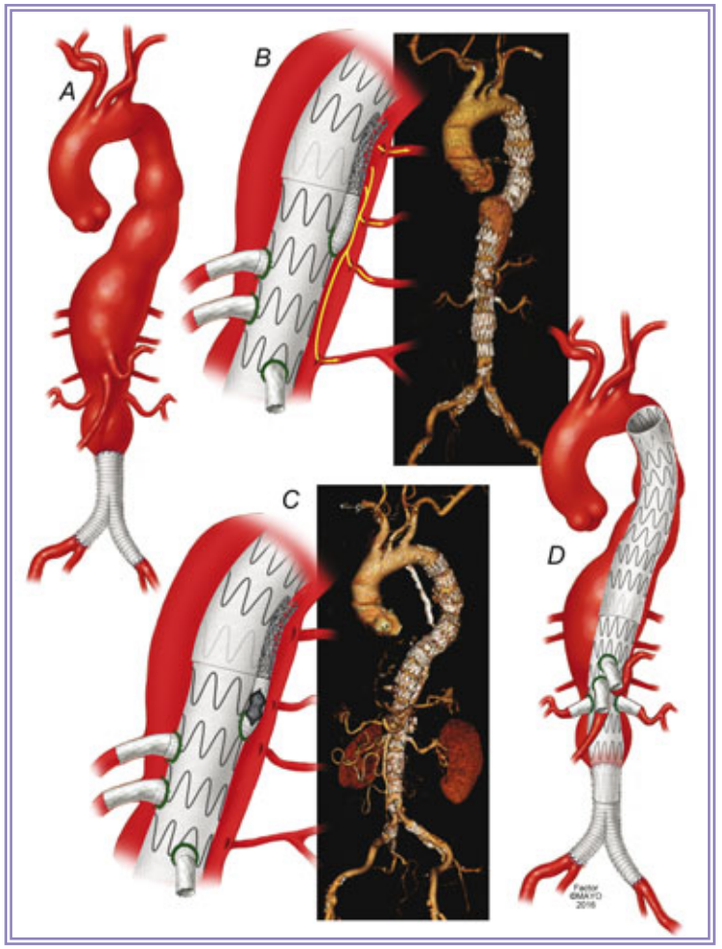

Fig. 37.6 Illustration of a patient with extent II thoraco-abdominal aortic aneurysm (**A**) and prior infra-renal aortic repair. The patient was treated by four-vessel branched endograft with a temporary perfusion branch (**B**). The perfusion branch was occluded 1 week later using Amplatzer plug (**C**) with resolution of the intentional endoleak (**D**). By permission of Mayo Foundation for Medical Education and Research. All rights reserved.

37.5 Intra-operative maneuvers

Our protocol uses neuromonitoring to trigger intra-operative maneuvers to optimize spinal cord perfusion. A decrease in MEP/SSEPs triggers introduction of sequential standardized maneuvers as depicted in Figure 37.7. These maneuvers include incremental changes in MAP and CSF pressure as previously described. The MAP goals are raised from 80 mmHg up to 100 mmHg with norepinephrine and/ or vasopressin infusion along with simultaneous decrease in CSF drain pressure from 10 mmHg to 5 or 0 mmHg.

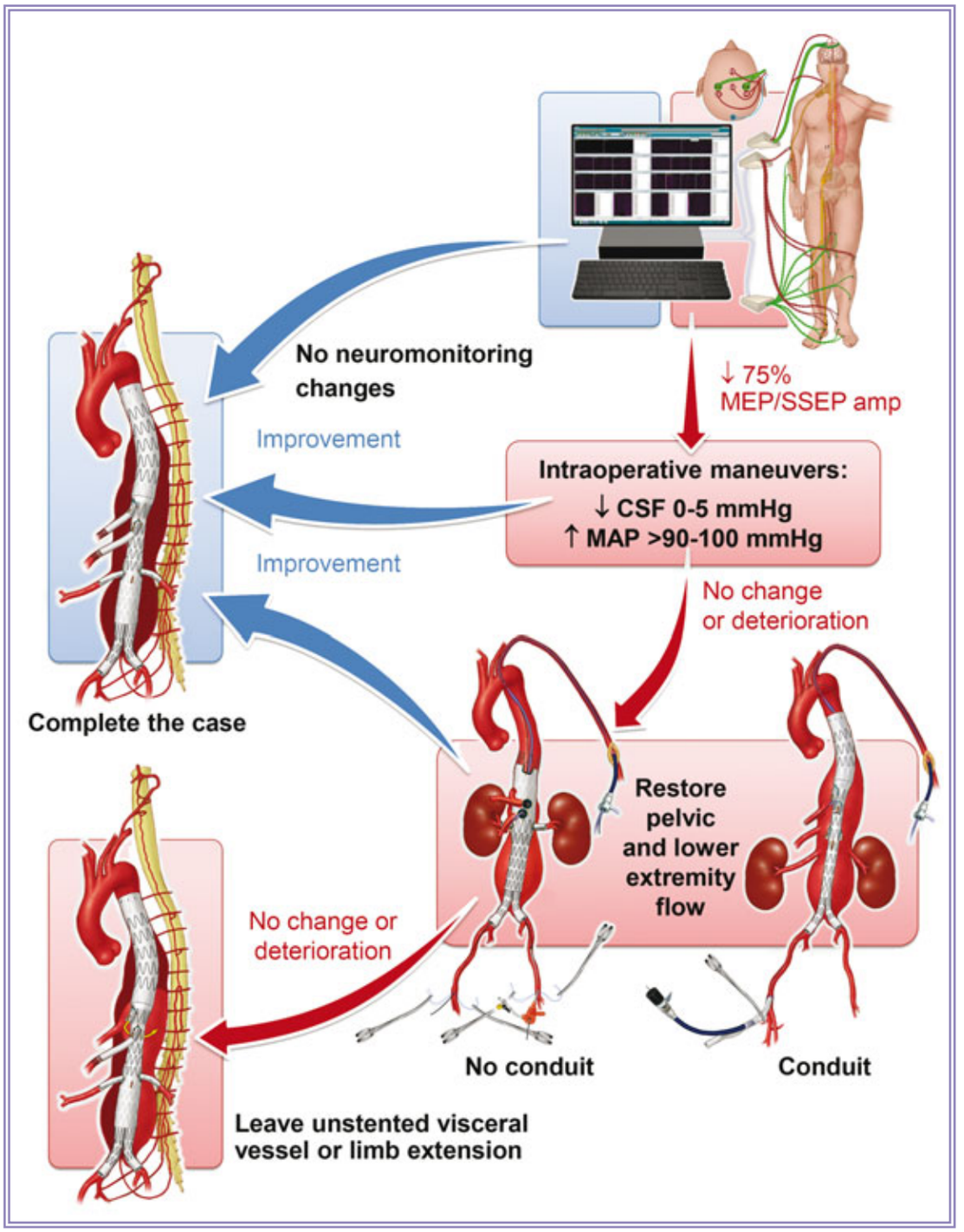

Fig. 37.7 Standardized maneuvers and protocol triggered by changes in motor and somatosensory-evoked potentials. By permission of Mayo Foundation for Medical Education and Research. All rights reserved.

Response to these maneuvers is recorded and the MAP and CSF pressure parameters are readjusted accordingly. In patients with improvement after maneuvers, the procedure is completed in standard fashion. In those with no change or deterioration in neuromonitoring, flow is restored to the pelvis and LEs as fast as possible by changing the sequence of target vessel stenting as depicted in Figure 37.7. In patients with normalization of MEP/SSEPs after lower extremity flow is restored, the procedure is completed. If changes persist, the procedure is left incomplete with flow into the sac via the celiac branch or contra-lateral iliac limb whenever possible.

37.6 Peri-operative management

All patients are closely monitored following the procedure in a closed cardiovascular intensive care unit by a dedicated critical care team. Spinal fluid pressure is set at a baseline of 10 mmHg as previously described. The spinal drain is opened for 15 min every hour with a maximum drainage of 20 mL per hour, after which the drain is clamped for the remaining of the hour. Similar adjunctive maneuvers are utilized in the post-operative setting if neurological changes are observed on physical examination. MAP goals are incrementally raised up to 100 mmHg. In addition, transfusion of blood products is performed to keep a target hemoglobin ≥10 mg/dL and normal coagulation profile should the need to reposition or replace a spinal drain be required. CSF pressure is decreased to 5 or 0 mmHg. CSF pressure is raised to 10 mmHg once neuromonitoring or examination improves.

37.7 Results

This protocol has been studied prospectively in our institution with a total of 232 patients enrolled to date. SCI prevention protocol was indicated in 166 of 232 patients (72%) treated by F-BEVAR for pararenal aneurysms in 22 patients, Extent IV TAAAs in 59 and Extent I-III TAAAs in 85 (Fig. 37.8). CSF drainage was successful in 162 patients (98%) and stable

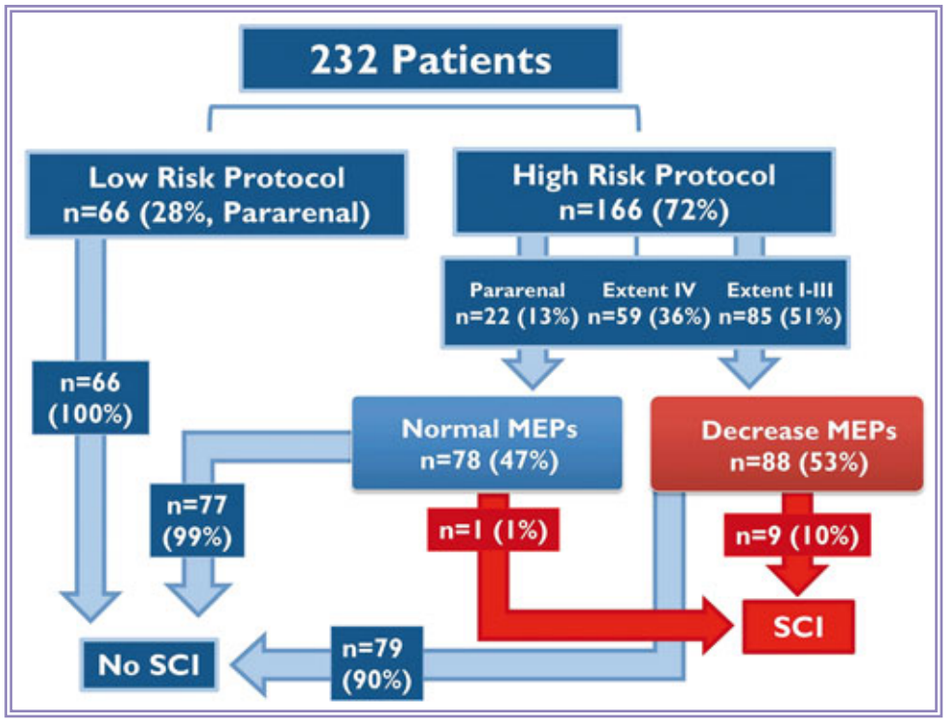

Fig. 37.8 Risk stratification, changes in neuromonitoring and incidence of spinal cord injury of 232 patients treated by fenestrated-branched endovascular aortic repair.

Table 37.2 Timing, peak and recovery of the spinal cordy injury in 232 patients treated by fenestrated-branched endovascular aortic repair

Case	Aneurysm extent	Changes in MEP/SSEP	Timing Grade[a] of SCI	Peak Grade of SCI	Follow-up Grade of SCI	Mechanism
1	Pararenal	Yes	Immediate Grade 3a	2 days Grade 3c	189 days Grade 2	Embolic
2	Extent III	Yes	Delay (9 days) Grade 1	4 days Grade 2	6 days Grade 0	Hypoflow
3	Extent II	Yes	Immediate Grade 3a	3 day Grade 3c	337 days Grade 0	Hypoflow
4	Extent III	No	Delay (3 days) Grade 3a	Same day Grade 3a	17 days Grade 0	Compression Spinal hematoma
5	Extent III	Yes	Delay (3 days) Grade 1	1 day Grade 2	16 days Grade 0	Compression Spinal hematoma
6	Extent II	Yes	Immediate Grade 3c	Same day Grade 3c	46 days Grade 2	Embolic
7	Extent II	Yes	Immediate Grade 3c	Same day Grade 3c	22 days Grade 0	Hypoflow
8	Extent II	Yes	Delay (1 day) Grade 2	Same day Grade 2	1 day Grade 0	Hypoflow
9	Extent II	Yes	Delay (1day) Grade 2	Same day Grade 2	6 day Grade 0	Hypoflow
10	Extent II	Yes	Delay (27 days) Grade 2	1 day Grade 3a	15 days Grade 0	Hypoflow

MEP, motor evoked potential; SSEP, somatosensory evoked potential; SCI, spinal cordy injury; a Grading system proposed by the SVS TEVAR reporting standards [35].

neuromonitoring was achieved in all patients. Eighty-eight patients (53%) had changes in neuromonitoring starting 50 ± 37 minutes after introduction of the aortic device. Changes in neuromonitoring improved with maneuvers in all except for 10 patients (11%) who had persistent decline in MEP/SSEPs after low limb reperfusion. All 10 patients had TASP by leaving a renal-mesenteric branch or contralateral iliac gate incomplete. There was one 30-day or in-hospital mortality (0.4%) in the cohort. The cause of death was subarachnoid hemorrhage from CSF drainage. Ten patients (4%) developed SCIs, including 6 paraplegia and 4 paraparesis (Table 37.2). SCIs were immediate in 4 and delayed in 6 with an incidence of 1% for pararenal, 0% for Extent IV, 13% for Extent III and 10% for Extent I-II TAAAs. The probable cause of SCI was hemodynamic compromise in 6 patients, embolic in 2 and epidural hematoma in 2. SCIs occurred in 1/78 patients (1%) with normal neuromonitoring and in 9/88 patients (10%) who had decline in MEP/SSEPs (P = 0.02). Among the 10 patients with TASP, neurologic exam was normal in 8 and showed SCI in 2. TASP closure was completed in all patients at 22 ± 16 days, with one SCI 2 days after completion. All 3 patients with post-TASP SCI had complete recovery to ambulatory status. Overall, 2 patients (1%) had permanent paraplegia, which was immediate and probably embolic in both. Factors associated with SCI included extent I-III TAAAs, change in neuromonitoring and need for TASP.

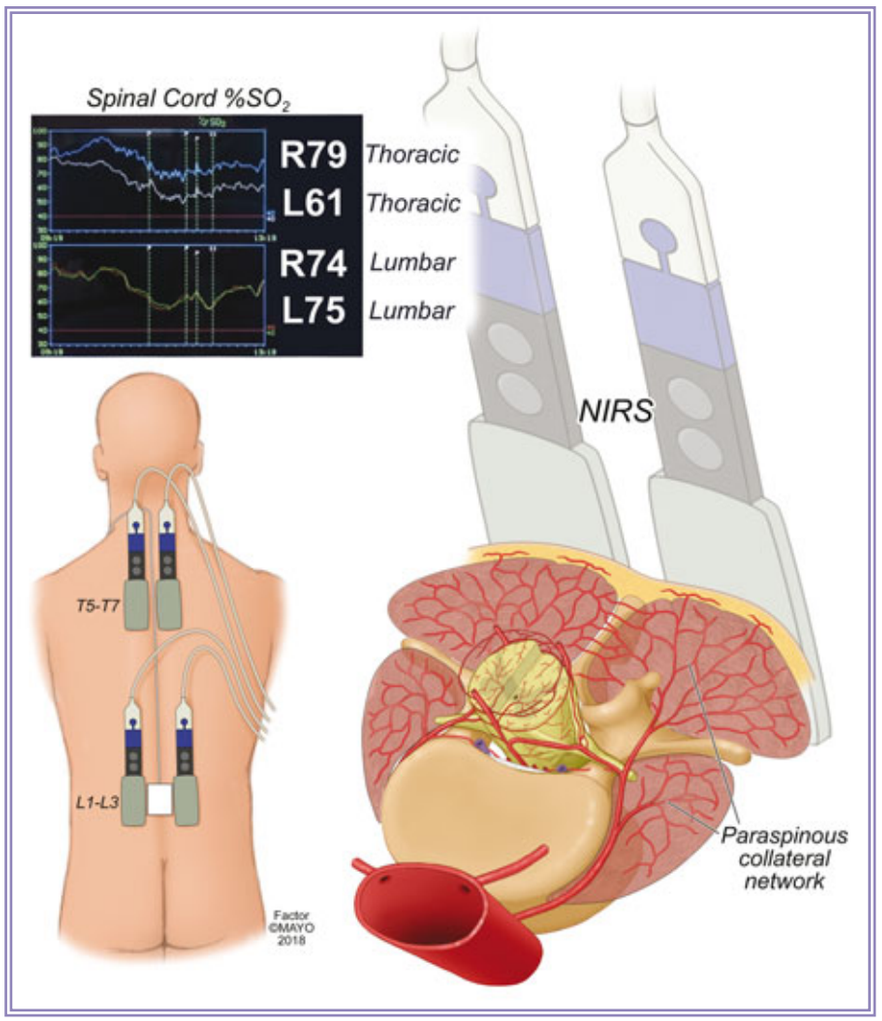

Fig. 37.9 Preoperative setup for neuromonitoring using NIRS during F-BEVAR: CSF drainage and bilaterally placed NIRS optodes at the thoracic (T5-T7) and lumbar (L1-L3) levels. Monitoring of the PCN by NIRS: oxygenation of the paraspinous vasculature – fed by the aortic segmental arteries – may directly correlate with spinal cord blood supply. By permission of Mayo Foundation for Medical Education and Research. All rights reserved.

37.8 Near-infrared spectroscopy

Near-infrared spectrascopy (NIRS) monitoring of hemoglobin oxygen saturation in the paraspinous muscles has recently been shown to be a feasible continuous non-invasive technique to assess the adequacy of collateral network perfusion (Fig. 37.9). Feasibility studies have shown that its use reliably predicts patients who develop SCI and correlates well with MEP results. Possible advantages of NIRS are is that anesthetic or peripheral ischemia does not interfere with measurement and NIRS measurements can be easily performed in the postoperative period [32, 33]. However, further research is required to prove reproducibility, determine optimal optode positioning and indicate whether its use can reduce the risk of SCI.

37.9 Conclusion

Spinal cord ischemia is a devastating complication of aortic surgery and remains so in the endovascular era. Many approaches have been described to help mitigate these risks, some of which are applicable to endovascular surgeries. In both experimental models and in clinical scenarios, staging of repairs of TAAA repair appears to provide a protective advantage against the development of SCI. In addition, meticulous attention to the perioperative management

with using neuromonitoring intraoperatively of these patients may help prevent this complication or at least limit the severity with which it occurs. Additional investigations are necessary in order to augment current treatment plans in order to further limit the development of SCI. Regardless, additional help is necessary in order to make thoraco-abdominal aortic aneurysm repair safer and more effective.

REFERENCES

1) Greenberg RK, Lu Q, Roselli EE, et al. Contemporary analysis of descending thoracic and thoracoabdominal aneurysm repair: a comparison of endovascular and open techniques. Circulation 2008; 118(8): 808-17.
2) Eagleton MJ, Shah S, Petkosevek D, Mastracci TM, Greenberg RK. Hypogastric and subclavian artery patency affects onset and recovery of spinal cord ischemia associated with aortic endografting. J Vasc Surg 2014; 59(1): 89-94.
3) Safi HJ, Estrera AL, Miller CC, et al. Evolution of risk for neurologic deficit after descending and thoracoabdominal aortic repair. Ann Thorac Surg 2005; 80(6): 2173-9; discussion 9.
4) Coselli JS, Bozinovski J, LeMaire SA. Open surgical repair of 2286 thoracoabdominal aortic aneurysms. Ann Thorac Surg 2007;83(2): S862-4; discussion S90-2.
5) Maurel B, Delclaux N, Sobocinski J, et al. The impact of early pelvic and lower limb reperfusion and attentive peri-operative management on the incidence of spinal cord ischemia during thoracoabdominal aortic aneurysm endovascular repair. Eur J Vasc Endovasc Surg 2015; 49(3): 248-54.
6) Svensson LG, Crawford ES, Hess KR, Coselli JS, Safi HJ. Experience with 1509 patients undergoing thoracoabdominal aortic operations. J Vasc Surg 1993; 17(2): 357-68; discussion 68-70.
7) Tanaka H, Minatoya K, Matsuda H, et al. Embolism is emerging as a major cause of spinal cord injury after descending and thoracoabdominal aortic repair with a contemporary approach: magnetic resonance findings of spinal cord injury. Interact Cardiovasc Thorac Surg 2014; 19(2): 205-10.
8) Etz D, Luehr M, Aspern K, et al. Spinal cord ischemia in open and endovascular thoracoabdominal aortic aneurysm repair: new concepts. J Cardiovas Surg 2014; 55(2 Suppl 1): 159-68.
9) Etz CD, Weigang E, Hartert M, et al. Contemporary spinal cord protection during thoracic and thoracoabdominal aortic surgery and endovascular aortic repair: a position paper of the vascular domain of the European Association for Cardio-Thoracic Surgery. Eur J Cardiothorac Surg 2015; 47(6): 943-57.
10) O'Callaghan A, Mastracci TM, Eagleton MJ. Staged endovascular repair of thoracoabdominal aortic aneurysms limits incidence and severity of spinal cord ischemia. J Vasc Surg 2015; 61(2): 347-54. e1.
11) Feezor RJ, Martin TD, Hess PJ, Jr., et al. Extent of aortic coverage and incidence of spinal cord ischemia after thoracic endovascular aneurysm repair. Ann Thorac Surg 2008; 86(6): 1809-14; discussion 14.
12) Bisdas T, Panuccio G, Sugimoto M, Torsello G, Austermann M. Risk factors for spinal cord ischemia after endovascular repair of thoracoabdominal aortic aneurysms. J Vasc Surg 2015; 61(6): 1408-16.
13) Dias NV, Sonesson B, Kristmundsson T, Holm H, Resch T. Short-term outcome of spinal cord ischemia after endovascular repair of thoracoabdominal aortic aneurysms. Eur J Vasc Endovasc Surg 2015; 49(4): 403-9.
14) Scali ST, Wang SK, Feezor RJ, et al. Preoperative prediction of spinal cord ischemia after thoracic endovascular aortic repair. J Vasc Surg 2014; 60(6): 1481-90 e1.
15) Zipfel B, Buz S, Redlin M, et al. Spinal cord ischemia after thoracic stent-grafting: causes apart from intercostal artery coverage. Ann Thorac Surg 2013; 96(1): 31-8.
16) Estrera AL, Miller CC, 3rd, Huynh TT, et al. Preoperative and operative predictors of delayed neurologic deficit following repair of thoracoabdominal aortic aneurysm. J Thorac Cardiovasc Surg 2003; 126(5): 1288-94.
17) Guillou M, Bianchini A, Sobocinski J, et al. Endovascular treatment of thoracoabdominal aortic aneurysms. J Vasc Surg 2012; 56(1): 65-73.
18) Hanna JM, Andersen ND, Aziz H, et al. Results with selective preoperative lumbar drain placement for thoracic endovascular aortic repair. Ann Thorac Surg 2013; 95(6): 1968-74; discussion 74-5.
19) Katsargyris A, Oikonomou K, Kouvelos G, et al. Spinal cord ischemia after endovascular repair of thoracoabdominal aortic aneurysms with fenestrated and branched stent grafts. J Vasc Surg 2015; 62(6): 1450-6.
20) Bischoff MS, Scheumann J, Brenner RM, et al. Staged approach prevents spinal cord injury in hybrid surgical-endovascular thoracoabdominal aortic aneurysm repair: an experimental model. Ann Thor Surg 2011; 92(1): 138-46.
21) Etz CD, Kari FA, Mueller CS, et al. The collateral network concept: remodeling of the arterial collateral network after experimental segmental artery sacrifice. J Thorac Cardiovasc Surg 2011; 141(4): 1029-36.

22) Bischoff MS, Brenner RM, Scheumann J, et al. Staged approach for spinal cord protection in hybrid thoracoabdominal aortic aneurysm repair. Ann Cardiothor Surg 2012; 1(3): 325.
23) Achouh PE, Estrera AL, Miller III CC, et al. Role of somatosensory evoked potentials in predicting outcome during thoracoabdominal aortic repair. Ann Cardiothorac Surg 2007; 84(3): 782-8.
24) Jacobs MJ, Meylaerts SA, de Haan P, Bas A, Kalkman CJ. Strategies to prevent neurologic deficit based on motor-evoked potentials in type I and II thoracoabdominal aortic aneurysm repair. J Vasc Surg 1999; 29(1): 48-59.
25) Banga PV, Oderich GS, Reis de Souza L, et al. Neuromonitoring, cerebrospinal fluid drainage, and selective use of iliofemoral conduits to minimize risk of spinal cord injury during complex endovascular aortic repair. J Endovasc Ther 2016; 23(1): 139-49.
26) Sobel JD, Vartanian SM, Gasper WJ, et al. Lower extremity weakness after endovascular aneurysm repair with multibranched thoracoabdominal stent grafts. J Vasc Surg 2015; 61(3): 623-8.
27) Harrison SC, Agu O, Harris PL, Ivancev K. Elective sac perfusion to reduce the risk of neurologic events following endovascular repair of thoracoabdominal aneurysms. J Vasc Surg 2012; 55(4): 1202-5.
28) Kasprzak PM, Gallis K, Cucuruz B, et al. Temporary aneurysm sac perfusion as an adjunct for prevention of spinal cord ischemia after branched endovascular repair of thoracoabdominal aneurysms. Eur J Vasc Endovasc Surg 2014; 48(3): 258-65.
29) Geisbusch S, Stefanovic A, Koruth JS, et al. Endovascular coil embolization of segmental arteries prevents paraplegia after subsequent thoracoabdominal aneurysm repair: an experimental model. J Thorac Cardiovasc Surg 2014; 147(1): 220-6.
30) Etz CD, Debus ES, Mohr FW, Kolbel T. First-in-man endovascular preconditioning of the paraspinal collateral network by segmental artery coil embolization to prevent ischemic spinal cord injury. J Thorac Cardiovasc Surg 2015; 149(4): 1074-9.
31) Luehr M, Salameh A, Haunschild J, et al. Minimally invasive segmental artery coil embolization for preconditioning of the spinal cord collateral network before one-stage descending and thoracoabdominal aneurysm repair. Innovations (Phila). 2014; 9(1): 60-5.
32) Etz CD, von Aspern K, Gudehus S, et al. Near-infrared spectroscopy monitoring of the collateral network prior to, during, and after thoracoabdominal aortic repair: a pilot study. Eur J Vasc Endovasc Surg 2013; 46(6): 651-6.
33) Boezeman RP, van Dongen EP, Morshuis WJ, et al. Spinal near-infrared spectroscopy measurements during and after thoracoabdominal aortic aneurysm repair: a pilot study. Ann Thorac Surg 2015; 99(4): 1267-74.
34) Drinkwater SL, Goebells A, Haydar A, et al. The incidence of spinal cord ischaemia following thoracic and thoracoabdominal aortic endovascular intervention. Eur J Vasc Endovasc Surg 2010; 40(6): 729-35.
35) Fillinger MF, Greenberg RK, McKinsey JF, Chaikof EL, Society for Vascular Surgery Ad Hoc Committee on TRS. Reporting standards for thoracic endovascular aortic repair (TEVAR). J Vasc Surg 2010; 52(4): 1022-33, 33 e15.

43

Aortoesophageal fistula following open and endovascular thoracic aortic repair: management strategies

Andrea L. Kahlberg, Alessandro Grandi, Diletta Loschi, Luca Bertoglio, Germano Melissano and Roberto Chiesa

43.1 Introduction

Aortoesophageal fistulae (AEF) are rare and, even when treatment is appropriately and timely performed, highly lethal. Aortoesophageal fistulae are most commonly found in association with thoracic trauma, aortic aneurysms, ruptured penetrating aortic ulcers, esophageal malignancies (primary fistulae), and as a complication of thoracic surgery, including aortic open or endovascular surgery in up to 1.7% of cases (secondary fistulae) [1, 2].

Open surgical repair of the thoracic aorta associated with esophageal reconstruction is traditionally considered the most radical treatment. Mortality rates of open surgery, however, are primarily due to hemorrhagic and septic complications, with rates reaching 61% in case of primary etiology and 78% in case of secondary fistulae [3, 4]. Although several alternative strategies have been reported in the literature, including extraanatomic bypass [5], and in-situ repair with cryopreserved homograft [6], there is a lack of consensus concerning the optimal treatment of AEF.

Thoracic endovascular aortic repair (TEVAR) has been proposed as an alternative strategy to surgical management of primary and secondary fistulae [7]. This technique, although less invasive, presents significant limitations in treating AEF, primarily the issue of graft contamination. A variety of combinations of TEVAR with surgical aortic repair, esophageal stent-grafting, esophageal reconstruction, mediastinal drainage, or even endoscopic use of fibrin glue at the level of the fistula were proposed by different authors [8, 9].

Today, with ever-growing numbers of interventional thoracic aortic procedures and increased follow-up periods, late complications of TEVAR have become increasingly evident, also including secondary AEF [10]. Relatively little is known about this pathology due to their rarity, the increased use of endovascular techniques, and the lack of multicenter reports [11, 12].

Andrea L. Kahlberg (kahlberg.andrea@hsr.it)
Vita-Salute San Raffaele University, Scientific Institute Ospedale San Raffaele, Milan, Italy

G. Melissano - R. Chiesa (eds), Aortic Complexities

43.2 Epidemiology and etiopathogenesis

Being first described by Dubrueil in 1818 [13], aortoesophageal (AEF) fistulae are well-known rare causes of massive gastrointestinal and respiratory bleeding, with high mortality rates. Primary aortoesophageal fistulae constitute less than 10% of all aortoenteric fistulae [1]. The most common pathogenic cause of AEF is thoracic aortic aneurysms (TAAs): the incidence of AEF following TAA rupture has been reported to range from 9.4 to 20.4% [14]. Dossa et al. found that 63% of AEF were the result of TAA, 16% were the consequence of oesophageal pathology (both benign and malignant) and 15% were caused by foreign body ingestion or prolonged nasogastric tube intubation [3].

Secondary AEF are defined as those that follow a previous operation and are a very severe complication of the procedure. Aortic fistulae into the oesophagus have been observed during infancy after patent ductus arteriosus or aortic coarctation repair; in adults they prevail after TAA treatment and have also been reported after repair of the aortic arch, thoracoabdominal aneurysms, type A and B dissection and Takayasu arteritis. Secondary AEF may occur as a complication of open or endovascular repair of the thoracic and thoracoabdominal aorta in 0.5 to 1.7% of patients [2, 15].

Erosion of the pulsating prosthetic material into the adjacent structure, graft infection with abscess rupture into the oesophagus or development of anastomotic pseudoaneurysm are the main mechanisms of secondary aortoenteric fistula formation after aortic surgical intervention. Anastomotic pseudoaneurysms following surgical repair of the thoracic aorta may arise from disruption of one or more arterial wall layers with extravasation of blood into surrounding spaces. Adjacent arteriosclerosis, fragility of the host tissue, local infection, disruption of the suture line, as well as inappropriately tight sutures, may all play a role in pseudoaneurysm development. Oesophagus compression by the pseudoaneurysm results in a local inflammatory response, formation of stable adhesions and tissue necrosis, leading to erosion and final fistulization. The formation of a fistula may also result from poor surgical technique if the suture needle passes through the thick oesophageal wall when the proximal aortic anastomosis is performed. This occurrence may be prevented by complete transection of the aorta prior to performing the anastomosis.

In cases of aortoesophageal fistula following endovascular repair, it has been suggested that the fistula may arise secondary to the development of pseudoaneurysm, endoleak into the residual aneurysm sac, or erosion of the stent-graft through the aorta by the rigid extremities located at either end of the stent [16]. Perforation usually occurs in a tortuous segment of the aorta, mainly in the isthmic region or in the middle third of the descending tract, where the vessel may curve, passing behind the oesophagus. In this respect, an increased weakness of the aortic wall at the level of stent-graft deployment, as in case of ruptured aneurysms, penetrating ulcers and trauma may contribute to the formation of a fistula. In addition, potential ischemic necrosis of the oesophageal wall due to stent-graft coverage of aortic side-branches that feed the oesophagus may be involved.

43.3 Diagnosis and natural history

The clinical presentation of AEF is generally referred to as Chiari's triad, consisting of midthoracic pain, sentinel hematemesis, and ultimately fatal exsanguination after an unpredictable symptom-free interval.

In a review of 500 cases of AEF, 59% had mid-thoracic pain, 45% experienced dysphagia, 65% had herald bleeding, and 45% showed Chiari's triad. The need for a high index of suspicion coupled with rapid evaluation is underscored by the fact that most cases of AEF are diagnosed postmortem (Fig. 43.1).

Besides fatal hemorrhage, complications of AEF include mediastinitis, infection of the surgical or endovascular graft, and sepsis [4].

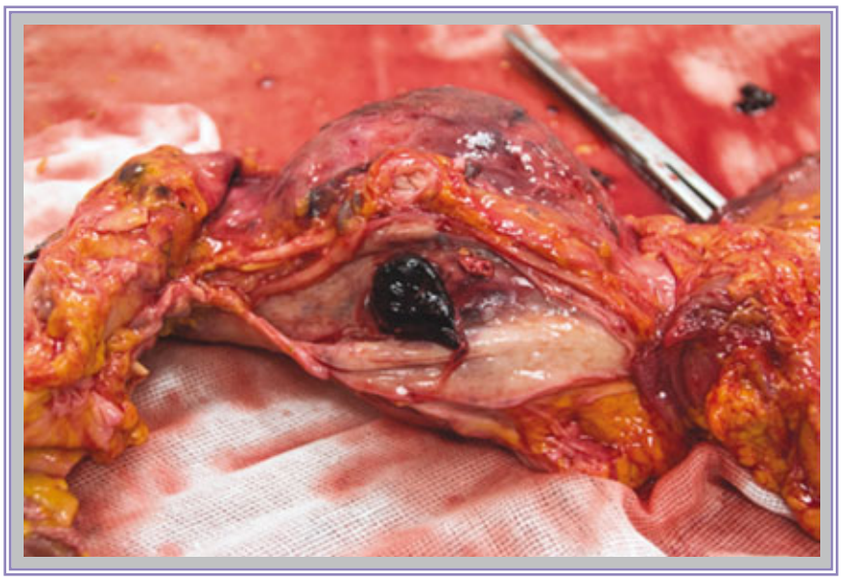

Fig. 43.1 Autoptic documentation of an aortic fistula with a clot plugging the lesion.

Except in patients presenting massive exsanguinating haemorrhage who require immediate emergency surgery, a number of investigative modalities can be used to confirm diagnosis and plan treatment. Oesophageal contrast studies can be helpful either by showing contrast material around the aortic prosthesis in patients with secondary AEF or by demonstrating oesophageal perforation thus drawing attention to the oesophagus (Fig. 43.2).

Esophagogastroduodenoscopy (EGD) may allow direct visualization of the prosthesis through oesophageal defects of variable size depending on the amount of tissue loss. Esopha-

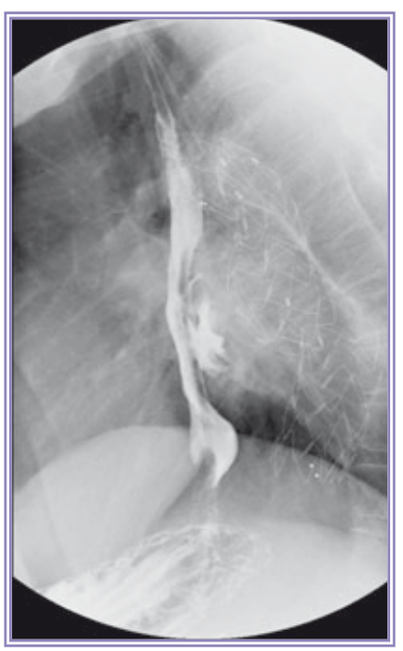

Fig. 43.2 Esophageal transit study showing contrast medium in the esophagus and expanding inside the aneurysm sac through the fistula.

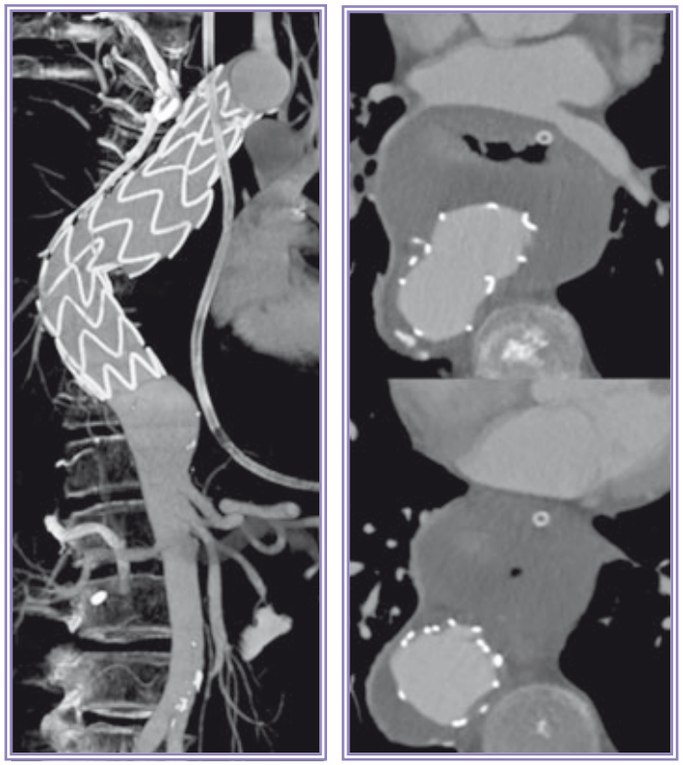

Fig. 43.3 CT scan showing the aneurysm sac in contact with the esophagus at the level of the previously deployed TEVAR, and the presence of periaortic air collection.

gogastroduodenoscopy can also show the presence of a variable amount of submucosal hematoma indicating extravasation of blood into the oesophageal wall. However even though some investigators advocate EGD as the diagnostic modality of choice for evaluation of AEF, others have advised against its use because several cases of fatal haemorrhage precipitated by flexible endoscopy have been reported [17]. However, dislodgment of occluding clots and traumatic rupture of an adherent vascular mass are potential serious complications. Some authors recommend that a surgical team be ready for emergency operation during the procedure.

Although arteriograms rarely reveal the fistula site, they can provide useful data for surgical planning. Computed tomography scanning is the most valuable diagnostic method. Findings include visualization of a false aneurysm between the oesophagus and aorta in patients presenting AEF induced by ingested foreign bodies and of adherences between the oesophagus and prosthesis in patients with secondary AEF. In a few cases computed tomography scanning allows visualization of gas effusion either around the prosthesis or inside an aneurysm (Fig. 43.3). Fluorodeoxyglucose (FDG)-positron emission tomography (PET) can be performed to have a complete picture in those cases in which the presence of an AEF is not clear and prosthesis infection is suspected.

Once the diagnosis is established, definitive treatment should be expeditious; if left untreated, AEF are invariably fatal. In a review of AEFs, Coselli and Crawford reported that more than 60% of patients presenting with a herald gastrointestinal bleed die within 6 hours [18].

43.4 Surgical treatment

It is well known that AEF is a great surgical challenge for the vascular specialist. Three main issues need to be addressed: first, emergent aortic repair to prevent exsanguination; second, extensive debridement of the arterial bed and arterial reconstruction; third, esophageal repair. It is quite clear that this kind of approach would be the best option for a definitive treatment,

but unfortunately only a small percentage of patients are considered fit for this major surgery with acceptable operative risk. Access to the aorta is often particularly demanding due to the presence of vast adhesions in patients with previous thoracic interventions, as is the case of secondary fistulae, with a high risk of adjacent organ lesions and significant blood loss.

Direct, simple aortic suture or patch angioplasty are rarely employed, almost only for the treatment of small lesions associated with foreign bodies ingestion [19]. Aortic replacement is usually required for larger lesions. In most cases, a variety of possible alternatives can be applied, such as *in situ* reconstructions, extra-anatomic bypasses, or temporary solutions such as drainages or endovascular procedures, performed in order to stabilize the patient before a definitive vascular reconstruction.

43.4.1 In situ reconstruction: aortic homografts

In case of fistulae involving the thoracic aorta, intrinsic anatomic limitations often mandate for *in situ* reconstruction. The use of cryopreserved aortic homografts relies on the fact that they are assumed to be infection resistant and it is derived from cardiac surgery where they are extensively used for acute aortic valve endocarditis [20]. In vascular surgery the main experience in the use of homografts comes from the treatment of AEF and ABF. Some authors also advocate the use of fresh tissue allografts, preserved at 4 °C [6]. The major clinical role for these conduits seems to be in replacement of the thoracic and suprarenal aorta.

With minor differences in each country, homografts are harvested from heart-beating organ donors who fulfill criteria for cryopreserved heart valve homografts. After antimicrobial decontamination and extensive microbiology tests, the homografts are frozen and preserved to – 180 °C. Only great vessels with a warm ischemia time less than 6 hours are collected.

The use of allografts, both fresh or frozen, may still face some problems, the most important concern being their resistance to infection. In particular, homografts show a low resistance to necrotizing micro-organisms such as *Pseudomonas aeruginosa* [21]. Moreover, little is known on their durability and stability, especially in terms of the development of calcification, thrombosis or aneurysm when used in the thoracic aorta.

On the other hand, homografts use in the abdominal aorta has so far given contrasting results. The United States cryopreserved aortic allograft registry for abdominal aortic reconstruction in infected fields has shown concerning graft-related complications in 56 patients treated for primary graft infection, mycotic aneurysm, and aortic graft-enteric erosion. Persistent infection with perianastomotic hemorrhage was present in 9% of cases, graft limb occlusion in 9% of cases and pseudoaneurysm in 2% [22].

The advocates of homograft use consider them as a safe alternative to prosthetic extraanatomic graft reconstruction, as they allow a much simpler *in situ* reconstruction with reduced periaortic debridement. They need a shorter perioperative antibiotic treatment and are associated to lower rates of reinfection [23]. Coselli et al. have shown a far better outcome, in terms of early mortality, in patients treated with an *in situ* reconstruction using cadaveric homografts compared to those receiving a prosthetic graft. They reported a 100% (2/2) mortality rate in patients with prosthetic grafts while a 0% (0/4) mortality in patients receiving a homografts [23].

So far, it is not clear why cryopreserved homografts may reduce risk of reinfection. The involvement of recipient immune cells, passive release of bacterial substances from the graft wall and chemical properties of the homograft itself could be speculative explanations [21].

Surgical reconstruction with the use of cryopreserved homografts requires approach to the descending aorta through a left sided posterolateral thoracotomy. The use of adjunct to reduce the ischemic time to the distal organ and to the spine is strictly recommended as surgical excision of the endograft and reconstruction can be extremely time consuming, due to the ongoing infection which can lead to periaortic inflammation and adhesions. Mild permissive hypothermia is also useful. During excision of the infected graft is important to try

to avoid damage to the left lung or to the esophagus with contamination of the posterior mediastinum.

Reimplantation of critical intercostals is rarely performed, unless they are involved in the distal neck area. The homograft is inserted in an end-to-end fashion and anastomoses are performed using a running polypropilene suture.

43.4.2 In situ reconstruction: antibiotic-impregnated and "silver-coated" prosthetic grafts

As an alternative to homografts, some authors have described the use of antibiotic-impregnated prosthetic grafts to treat infection of surgical aortic grafts [26]. The most extensively studied antibiotic-impregnated prostheses are rifampin-soaked grafts. These are prepared by simply soaking the graft in rifampin at a concentration of 60 mg/mL for 15' before implantation. It has been demonstrated that these grafts are able to release rifampin for at least 48 hours once the blood circulation is restored. In an attempt to improve resistance to infection, rifampin has also been mixed within the collagen during graft fabrication, augmenting the duration of the antibacterial activity.

A second type of graft with antibacterial activity is the so called "silver-coated" graft. This prosthesis has a layer of silver acetate lining the surface, in order to confer resistance to infection. The use of silver grafts has so far produced contrasting results and their ultimate role in term of resistance to infection is still to be defined [25]. More recently, triclosan (5-chloro-2-(2,4-dichlorophenoxy)phenol) has been added to silver, and the combination of these two anti-infective agents has demonstrated promising results in *in vitro* studies of grafts contaminated even with bacteria and fungi directly collected from infected aortic grafts retrieved from patients (Fig. 43.4) [26].

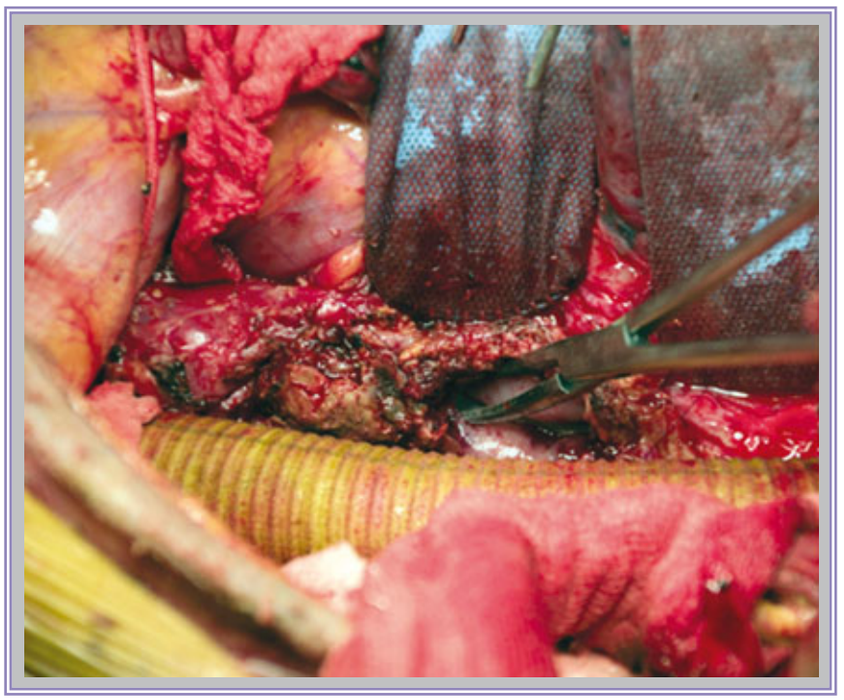

Fig. 43.4 Intraoperative photograph detailing a surgical open aortic replacement with a Silver-coated+Triclosan graft (Intergard Synergy, Maquet, Rastatt, Germany), and the presence of an associated esophageal lesion.

Even with all their limitations, the use of *in situ* reconstruction with antibiotic impregnated graft is of particular value in the setting of the thoracic aorta, as the use of extra-anatomic bypasses is usually technically demanding. Moreover, it is important to implement additional strategies to prevent reinfection of prosthetic grafts, in particular the use of viable tissue flaps (either omentum, pericardial fat pad, thymus, dorsal muscle flap, intercostal muscle, etc.) or biological materials is strongly advised to obliterate dead space, or, in the presence of a fistula, to reinforce the visceral wall [27]. Other adjuncts, such as perigraft catheters for postoperative antibiotic irrigation were also previously reported.

43.4.3 Extra-anatomic reconstructions

Instead of *in situ* reconstructions, extra-anatomic bypasses (EAB) may be performed to restore a vascular continuity far from the contaminated fields. The experience with EABs comes principally from the treatment of infected graft of the abdominal aorta where this technique, associated to ligation of the aorta and removal of the infected surgical graft, is often performed [5].

In the thoracic aortic district, the use of axillofemoral bypass as a stand-alone procedure is quite limited and it is described only in small case series for the treatment of aortic coarctation or re-coarctation in high-risk patients, or in other selected cases [28].

More often, the use of the axillobifemoral or axillofemoral configuration is reported as a bridge treatment to temporary relieve state of acute distal ischemia in unstable patients until they can be submitted to more extensive and durable reconstruction. This strategy is described in patients with acute partial thrombosis of a thoracic endograft or with thrombosis of a previous thoracic surgical graft to rapidly solve acute limb and visceral ischemia. All these patients are subsequently treated with a more complete revascularization, either in situ or with other extra-anatomic bypasses, such as ascending to celiac aortic bypass.

A problem noted with the axillofemoral graft is the development of refractory hypertension of the upper district, due to the restricted size of the graft [5]. An axillofemoral graft is not recommended as the only treatment in the thoracic aorta for different reasons. First of all, the resting flow of an axillobifemoral graft is approximately 600 mL/min [29]. Considering that the average flow in each femoral artery is 300-400 mL/min, it is self evident that this kind of configuration can hardly provide sufficient flow to both the viscera and the lower limbs. Furthermore, the risk of aortic stump blowout is extremely likely in the thoracic aorta.

The most used EAB configuration in case of thoracic aorta infection is the so called "ventral aorta". This surgical technique was initially described for treatment of patients with severe or recurrent aortic coarctation, but many authors have used it in different settings such as aortic aneurysms, mycotic aneurysms, infection of surgical thoracic or thoracoabdominal aortic grafts [30].

This management strategy is carried out as a two-step procedure. In the first step the EAB is performed, whereas the aortic fistula is addressed in the second step, usually with ligation of the thoracic aorta. The ligature of the aorta has to be carried out as distal as possible from the infected graft, in order to use healthy aortic wall to close the breach. Avoiding cardiopulmonary bypass, circulatory arrest and aortic cross-clamping are the main advantages of this approach, reducing the physiologic stress associated to these maneuvers [31].

In the first operation, an ascending to abdominal aorta conduit through an anterior approach is performed. In this case the ascending aorta is approached through a median sternotomy extending to an upper laparotomy incision. The selected bypass is usually a 16 to 24 mm Dacron graft, which is sutured proximally to the ascending aorta and distally to the supraceliac aorta. Anastomoses are completed using a side biting clamp after circumferential dissection of the vessel. The graft descends on the right side of the heart to the diaphragm. The right mediastinal pleura and the diaphragm are incised at the pericardiophrenic angle, where the graft enters the abdomen. In the abdomen, the graft is passed between diaphragm and the

left lobe of the liver to reach the supraceliac aorta. A key point in the abdominal step is to fully mobilize the left lobe of the liver through incision of the triangular ligament.

Many authors report good results with this technique, if compared to others (such as axillobifemoral or biaxillofemoral), with good long-term patency rates and optimal perfusion to the viscera and the lower limbs [32]. Moreover, the "ventral aorta" avoids the development of chronic and refractory hypertension. The durable patency of this bypass is due to the fact it has an inflow site from the ascending aorta which is at high flow. Main limitations are represented by patients presenting acute hemorrhage or cases of aortic arch or proximal descending aorta infections.

Its advantages are clear, as the new graft lies far from the infected field and the risk of immediate reinfection is quite reduced [31]. On the other hand, the fact that the new graft is placed in a patient who still has a potential source of bacteraemia (i.e. the infected stent graft) rises some concern on the risk of distal seeding and reinfection through the blood stream.

43.4.4 Endovascular treatment

Mortality rates of open surgery to treat AEF are still prohibitive, especially in case of massive gastrointestinal bleeding. Moreover, a considerable number of patients are not suitable to open surgery when the fistula is discovered, due to their poor general conditions. These patients are usually deemed to conservative treatment, resulting in death in almost all cases. For these reasons, an endovascular approach may be used as an alternative strategy to surgical management in case of hemorrhagic complications of AEF [33]. Although less invasive, TEVAR presents however important limitations, mainly related to the high risk of endograft contamination. Therefore, TEVAR has to be considered only as a first line emergent treatment, in order to obtain immediate control of aortic bleeding. In good surgical candidates, coverage of the aortic lesion, along with an aggressive antibiotic therapy, may be used to achieve an improvement in the patient's general conditions, serving as a "bridge" to open surgical treatment of the aortic and/or esophageal defect (Fig. 43.5).

Riesenman et al., published a case of a patient presenting with a thoracic endograft infection with a proximal and distal pseudoaneurysm; they first inserted two thoracic endografts to exclude the pseudoaneurysms and then, during a second procedure, they performed an EAB followed by removal of all the endograft components [31]. Otherwise, in the overtly moribund patient TEVAR has been proposed as the most appropriate definitive strategy, as a palliative procedure [33].

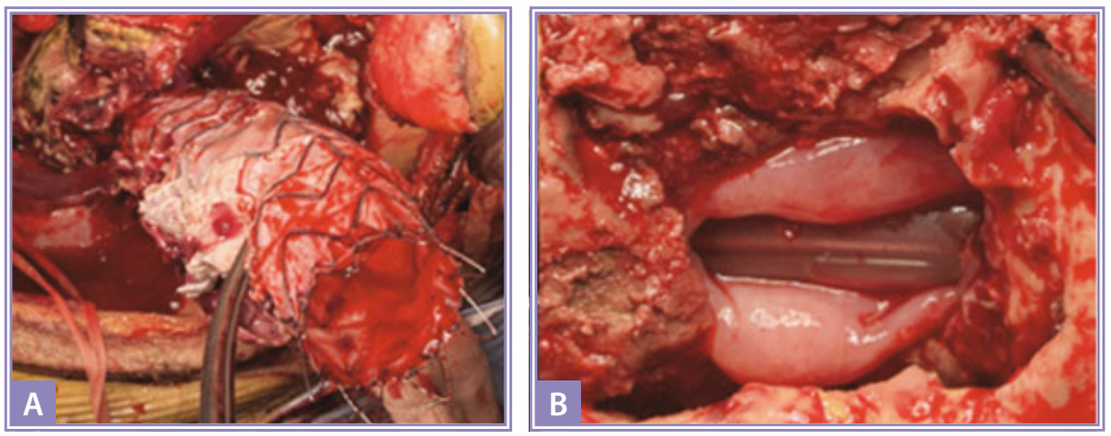

Fig. 43.5 Intraoperative pictures detailing (**A**) the removal of a previously deployed endograft and (**B**) an esophageal lesion where the naso-gastric tube can be clearly seen.

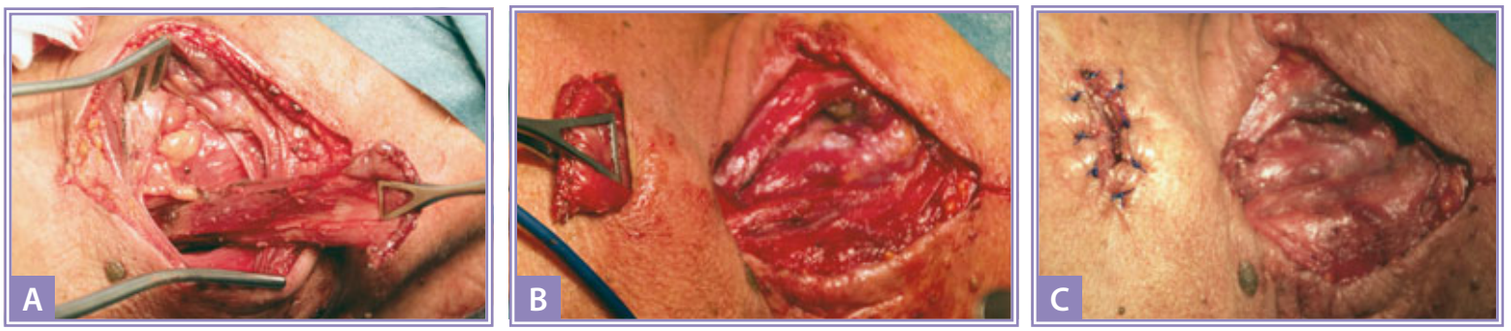

Fig. 43.6 Intraoperative pictures detailing the creation of an esophagostomy after esophageal stripping and gastrectomy. **A**) The proximal end of the esophagus is mobilized through a left cervicotomy, (**B**) it is then tunneled through the subcutaneous tissues and (**C**) then sutured to the skin.

43.4.5 Esophageal repair

In addition to repairing the aorta, the esophageal defect should be addressed. A small esophageal defect may be treated by direct repair and muscle flap interposition, whereas patients with a larger esophageal defect may undergo esophageal resection and subsequent reconstruction with gastric or colonic interposition [34]. Additional surgical maneuvers are usually required in case of esophageal partial or total resection, including nutritional gastrostomy or enterostomy, and cervical esophagostomy to ensure salivary drainage (Fig. 43.6).

43.5 Systematic review of the English literature

We performed an electronic health database search using the most important electronic search engines (PubMed, MEDLINE, EMBASE, Scopus, Google Scholar, Ovid, and the Cochrane Library), identifying articles that were published from 1997 to 2017 reporting single-center experience on descending thoracic aortic surgical graft or endograft infection, with or without a known associated fistula. Ascending aortic grafts were excluded, whereas thoracoabdominal infected grafts were included in the analysis.

From an initial pool of 1657 retrieved articles, 39 single-center studies met all eligibility criteria, including 126 patients with infected surgical grafts (49 patients) or infected endografts (77 patients).

Association with AEF was significantly less common with surgical grafts than with endografts (31% vs 60%, respectively; $P = 0.01$). In surgical grafts, mean time interval from index procedure to diagnosis of infection was 966 days (approximately 32.2 months), significantly longer as compared to endografts (17.1 months; $P = 0.03$). As regards symptoms at presentation, pain and fever/chills were significantly more common with surgical grafts (73 vs 35%, $P < 0.001$; and 84 vs 61%, $P = 0.02$, respectively), whereas haematemesis was significantly more common with endografts (42 vs 19%, $P = 0.02$).

Conservative treatment mainly represented by antibiotic therapy sometimes associated with percutaneous drainage of fluid collections or flushing, was performed in 2% of patients with surgical grafts (1/49) and 18% with endografts (14/77). This approach resulted in a mortality rate of 100% at 30 days in surgical graft infection group, and 38%, 75%, 100% in endograft infection group at 30 days, 1 year, and 5 years, respectively.

In the remaining patients, different surgical strategies were used. Esophageal or respiratory fistula repair, without associated aortic repair, was performed in 1 patient with surgical graft infection (2%) and in 10 patients with endograft infection (13%). Surgical aortic repair associated to additional manoeuvres to repair the fistula was performed only in 2 patients with en-

dograft infection (3%), whereas surgical aortic repair without additional manoeuvres to repair the fistula was performed in 2 patients with surgical grafts (4%) and 7 with endografts (9%).

Endovascular aortic repair (TEVAR) was the most used approach. TEVAR associated to additional manoeuvres to repair the fistula was performed in 12 patients with surgical graft infection (27%) and in 23 patients with endograft infection (30%). TEVAR without additional manoeuvres to repair the fistula was performed in 25 patients with surgical graft infection (52%) and in 16 patients with endograft infection (21%).

Finally, temporary TEVAR to obtain hemodynamic stability followed by planned open surgical aortic repair was performed in 6 patients with surgical graft infection (12%) and in 5 patients with endograft infection (6%).

When pooling together results from published comparative studies in an odds-ratio meta-analysis, a trend of lower mortality rate at 1 year was found in patients with endograft infection as compared to patients with surgical graft infection (pooled OR = 0.3; 95% CI 0.9-14.7; p = 0.073).

Also, a trend toward better outcome was found in patients with surgical treatment as compared to conservative treatment (pooled OR = 0.6; 95% CI 0.1-2.3; p = 0.425), especially in case of infected graft explantation as compared to graft preservation (pooled OR = 0.3; 95% CI 0.1-1.0; p = 0.056).

43.6 Conclusions

AEF represent a not negligible late complication of both surgical repair of the thoracic aorta and of TEVAR. Treatment is always demanding and multidisciplinary. Conservative strategies alone, using antibiotics and minor surgical maneuvers (e.g. percutaneous drainage), seem to result invariably in patient's death.

Radical open surgical repair, addressing both the aortic and esophageal lesions, even if considered the most definitive strategy, is associated with high perioperative mortality rates and is often not feasible in urgent settings.

TEVAR has a predominant role in controlling the massive hemorrhage associated with AEF in emergency. In many reported cases, TEVAR was performed in association with surgical repair of the esophageal defect, and a definitive aortic surgical replacement was indefinitely postponed or abandoned. In other cases, after initial patient's stabilization, definitive aortic repair with infected graft explantation was accomplished, appearing the most effective and durable strategy.

REFERENCES

1) Hollander JE, Quick G. Aortoesophageal fistula: a comprehensive review of the literature. Am J Med 1991; 91(3): 279-87.
2) Lawrie GM, Earle N, De Bakey ME. Evolution of surgical techniques for aneurysms of the descending thoracic aorta: twenty-nine years experience with 659 patients. J Card Surg 1994; 9(6): 648-61.
3) Dossa CD, Pipinos II, Shepard AD, Ernst CB. Primary aortoenteric fistula: Part II. Primary aortoesophageal fistula. Ann Vasc Surg 1994; 8(2): 207.
4) Pipinos II, Reddy DJ. Secondary aortoesophageal fistulae. Ann Vasc Surg 1999; 13(6): 649-52.
5) Madan AK, Santora TA, Disesa VJ. Extra-anatomic bypass grafting for aortoesophageal fistula: a logical operation. J Vasc Surg 2000; 32(5): 1030-3.
6) Kieffer E, Chiche L, Gomes D. Aortoesophageal fistula: value of in situ aortic allograft replacement. Ann Surg 2003; 238(2): 283-90.
7) Taylor BJ, Stewart D, West P, Dunn JT, Cisek P. Endovascular repair of a secondary aortoesophageal fistula: a case report and review of the literature. Ann Vasc Surg 2007; 21(2): 167-71.
8) Civilini E, Bertoglio L, Melissano G, Chiesa R. Aortic and esophageal endografting for secondary aortoenteric fistula. Eur J Vasc Endovasc Surg 2008; 36(3): 297-9.

9) Mok VW, Ting AC, Law S, et al. Combined endovascular stent grafting and endoscopic injection of fibrin sealant for aortoenteric fistula complicating esophagectomy. J Vasc Surg 2004; 40(6): 1234-7.
10) Eggebrecht H, Mehta RH, Dechene A, et al. Aortoesophageal fistula after thoracic aortic stent-graft placement: a rare but catastrophic complication of a novel emerging technique. JACC Cardiovasc Interv 2009; 2(6): 570-6.
11) Chiesa R, Melissano G, Marone EM, et al. Endovascular treatment of aortoesophageal and aortobronchial fistulae. J Vasc Surg 2010; 51(5): 1195-202.
12) Chiesa R, Melissano G, Marone EM, Marrocco-Trischitta MM, Kahlberg A. Aortoesophageal and aortobronchial fistulae following thoracic endovascular aortic repair: a national survey. Eur J Vasc Endovasc Surg 2010; 39(3): 273-9.
13) Dubrueil O. Observations sur la perforation de l'esophage et de l'aorte thoracique par une portion d'os avale: avec de reflexions. J Univ Sci Med 1818; 9: 357-63.
14) Hooper WL. Haematemesis and melena due to rupture of a saccular aneurysm of the aorta into the ooesophagus. Postgrad Med J 1962; 38: 297-301.
15) Svensson LG, Crawford ES, Hess KR, Coselli JS, Safi HJ. Experience with 1509 patients undergoing thoracoabdominal aortic operations. J Vasc Surg 1993; 17(2): 357-68.
16) Hance KA, Hsu J, Eskew T, et al. Secondary aortoesophageal fistula after endoluminal exclusion because of thoracic aortic transection. J Vasc Surg 2003; 37: 886-8.
17) Benson MJ, Rouse D, van Someren N, et al. Fatal hemorrhage from an aorto-esophageal fistula precipitated by flexible endoscopy. Gastrointest Endosc 1991; 37: 193-6.
18) Coselli JS, Crawford ES. Primary aortoesophageal fistula from aortic aneurysm: successful surgical treatment by use of omental pedicle graft. J Vasc Surg 1990; 12(3): 269-77.
19) Wu MH, Lai WW. Aortoesophageal fistula induced by foreign bodies. Ann Thorac Surg 1992; 54(1): 155-6.
20) Camacho MT, Cosgrove DM. Homografts in treatment of prosthetic valve endocarditis. In: Loop FD (ed.). Seminars in thoracic and cardiovascular surgery. Philadelphia, PA: W.B. Saunders, 1995; pag. 32-7.
21) Koskas F, Goëau-Brissonnière O, Nicolas MH, Bacourt F, Kieffer E. Arteries from human beings are less infectible by Staphylococcus aureus than polytetrafluoroethylene in an aortic dog model. J Vasc Surg 1996; 23(3): 472-6.
22) Noel AA, Gloviczki P, Cherry KJ Jr, et al; United States cryopreserved aortic allograft registry. Abdominal aortic reconstruction in infected fields: early results of the United States cryopreserved aortic allograft registry. J Vasc Surg 2002; 35(5): 847-52.
23) Coselli JS, Köksoy C, LeMaire SA. Management of thoracic aortic graft infections. Ann Thorac Surg 1999; 67(6): 1990-3; discussion 1997-8.
24) Bandyk DF, Novotney ML, Johnson BL, Back MR, Roth SR. Use of rifampin-soaked gelatin-sealed polyester grafts for in situ treatment of primary aortic and vascular prosthetic infections. J Surg Res 2001; 95(1): 44-9.
25) Larena-Avellaneda A, Russmann S, Fein M, Debus ES. Prophylactic use of the silver-acetate-coated graft in arterial occlusive disease: a retrospective, comparative study. J Vasc Surg 2009; 50(4): 790-8.
26) Berard X, Stecken L, Pinaquy JB, et al. Comparison of the antimicrobial properties of silver impregnated vascular grafts with and without triclosan. Eur J Vasc Endovasc Surg 2016; 51(2): 285-92.
27) Heyer KS, Modi P, Morasch MD, et al. Secondary infections of thoracic and abdominal aortic endografts. J Vasc Interv Radiol 2009; 20(2): 173-9.
28) Connery CP, DeWeese JA, Eisenberg BK, Moss AJ. Treatment of aortic coarctation by axillofemoral bypass grafting in the high-risk patient. Ann Thorac Surg 1991; 52(6): 1281-4.
29) Schneider JR, McDaniel MD, Walsh DB. Axillofemoral bypass: outcome and hemodynamic results in high-risk patients. J Vasc Surg 1992; 15: 952-63.
30) Kieffer E, Petitjean C, Richard T, et al. Exclusion-bypass for aneurysms of the descending thoracic and thoracoabdominal aorta. Ann Vasc Surg 1986; 1(2): 182-95.
31) Riesenman PJ, Farber MA. Management of a thoracic endograft infection through an ascending to descending extra-anatomic aortic bypass and endograft explantation. J Vasc Surg 2010; 51(1): 207-9.
32) McKellar SH, Schaff HV, Dearani JA, et al. Intermediate-term results of ascending-descending posterior pericardial bypass of complex aortic coarctation. J Thorac Cardiovasc Surg 2007; 133(6): 1504-9.
33) Jonker FH, Heijmen R, Trimarchi S, et al. Acute management of aortobronchial and aortoesophageal fistulas using thoracic endovascular aortic repair. J Vasc Surg 2009; 50(5): 999-1004.
34) Flores J, Shiiya N, Kunihara T, Yoshimoto K, Yasuda K. Aortoesophageal fistula: alternatives of treatment case report and literature review. Ann Thorac Cardiovasc Surg 2004; 10(4): 241-6
35) da Silva ES, Tozzi FL, Otochi JP, et al. Aortoesophageal fistula caused by aneurysm of the thoracic aorta (successful surgical treatment, case report, and literature review). J Vasc Surg 1999; 30(6): 1150-7.